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14. ABSTRACT

Hostility and anger expression have been postulated as contributing factors to cardiovascular disease and have been associated with cardiovascular reactivity and stress-induced myocardial ischemia. The present research investigates relationships between hostility, anger expression, and defensiveness and cardiovascular measures of reactivity and ischemia. The study is an analysis of previously collected data from the Triggers of Myocardial Ischemia Study, in which participants underwent mental stress tasks (math, anger recall speech, and Stroop Color-Word) with concurrent assessments of cardiovascular reactivity and ischemia as well as brief emotional state assessments in the laboratory. Outside of the laboratory, a subset of these participants (n= 59) completed a set of questionnaires including the Marlowe-Crowne Scale of Social Desirability and the Cook-Medley Hostility Scale. Results indicated that higher Cook-Medley Hostility scores were positively correlated with anger expression changes following the math task (r=.308, p< .05), and Composite Hostility predicted anger expression changes to the same task (r=.334, p<.05). The Composite Hostility component Hostile Affect was a significant predictor of anger expression changes during all three tasks. Although hostility measures did not appear to be consistently predictive factors in stress-induced cardiovascular reactivity, changes in anger expression predicted systolic and diastolic blood pressure changes during the anger recall task, and marginally predicted heart rate changes to anger recall. Higher Total Hostility was marginally related to presence of ischemia during Anger Recall and Stroop tasks, but anger expression changes were not related to presence of ischemia. Hypothesized interaction effects between hostility and anger expression in eliciting cardiovascular reactivity and ischemia were not observed. Defensiveness, contrary to predictions, was not significantly negatively associated with anger expression changes, nor was it a significant predictor of cardiovascular reactivity or ischemia. The study suggests the importance of assessment of acute emotional states when assessing personality characteristics and their impact on measures of cardiovascular reactivity and ischemia. Study limitations include low power in the evaluation of factors related to myocardial ischemia, and the study's use of a data set not specifically designed to test the present study hypotheses. Further investigation into anger expression in addition to personality traits may help in understanding factors contributing to cardiovascular reactivity and adverse coronary events.

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ABSTRACT

Title of Dissertation: Hostility and anger expression: behavioral and cardiovascular responses to mental stress among cardiovascular disease patients

Dana L. Tucker, Doctor of Philosophy, 2002

Dissertation directed by: David S. Krantz, Ph.D.,
Professor, Department of Medical and Clinical Psychology

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Hostility and anger expression:

behavioral and cardiovascular responses to mental stress

among cardiovascular disease patients

by

Dana L. Tucker

Dissertation submitted to the Faculty of the Department of Medical and Clinical Psychology Graduate Program of The Uniformed Services University of the Health Sciences in partial fulfillment of the requirements for the degree of Doctor of Philosophy, 2002

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The dissertation process, while eventually boiling down to a judgment of the quality of my work and understanding, involved a number of key players who contributed to the process, directly and indirectly.

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INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of death in the United States and as such is a major health risk for millions of American adults. Almost one fourth of the adult population has diagnosed CVD, among which 12 million individuals have coronary artery disease (CAD: U.S. Dept of Health, Education, and Welfare, 1979; Center for Disease Control and Prevention, 2000; American Heart Association, 2000). CAD develops via the process of atherosclerosis, in which the coronary arteries supplying blood to the cardiac, or myocardial, tissue become narrowed with fatty plaque deposits. This may result in ischemia, myocardial infarction, and sudden cardiac death.

Standard risk factors such as high cholesterol, hypertension, and smoking fail to fully predict CAD incidence, and thus researchers have focused on other possible risk factors to include social, psychological, psychophysiological and environmental factors and their possible interactions, such as acute emotional stress interacting with standard risk factors, or exacerbating existent coronary artery disease. Among these factors, personality and behavioral traits have been linked to CAD risk (Rozanski, Blumenthal, & Kaplan, 1999). Anger and hostility have been foremost among the personality and behavioral traits that have been linked to CAD, with early research stimulated in the field by the work of Rosenman and Friedman on the Type A Behavior Pattern (TABP; Friedman & Rosenman, 1959; 1974; Rosenman & Friedman, 1974). However, the results of research on the effects of hostility and anger on cardiovascular disease outcomes and/or parameters such as cardiovascular reactivity and ischemia have been inconsistent (Hemingway & Marmot, 1999; Kubzansky & Kawachi, 2000; O'Malley, Jones, Feuerstein, & Taylor, 2000). It is possible that some of the underlying assumptions

regarding the relationships between hostility and anger do not hold true, and further, that other factors, such as repression or suppression due to defensiveness mediate the effects of hostility and anger on cardiovascular disease (Mischel, 1973; Helmers et al., 1995; Burns, Evon, & Strain-Saloum, 1999; Mann & James, 1998).

The introduction will consider research conducted in these areas. First to be reviewed is the role of acute stress in cardiovascular disease, including both the relationship of cardiovascular reactivity to coronary artery disease, and the induction of ischemia in individuals with cardiovascular disease via acute stress. Next, the constructs of anger, hostility, and defensiveness/repression will each be reviewed in relation to cardiovascular disease in general, and, more specifically to cardiovascular reactivity and ischemia. Finally, a rationale and hypotheses are presented for a study that assessed, first, the associations between hostility and anger, then second, between hostility and anger and cardiovascular reactivity and ischemia, and third, the possible moderating effects of repression/defensiveness on relationships between hostility and anger and cardiovascular reactivity and ischemia.

Acute stress and cardiovascular disease

This section will review the relationship of acute stress to cardiovascular disease, with a focus on cardiovascular reactivity and ischemia.

Cardiovascular reactivity and coronary artery disease

This section will address cardiovascular reactivity, its link to coronary artery disease, methods used to elicit cardiovascular reactivity in the laboratory, and research on

the link between cardiovascular reactivity and CAD.

Cardiovascular reactivity has been studied as a measure of stress responding in both healthy individuals and among cardiovascular disease patients (e.g. Everson, Goldberg, Kaplan, Julkunen, & Salonen, 1998; Porter, Stone & Schwartz, 1999; Arrighi et al., 2000). It has been presented either as a marker of pathogenic processes in atherosclerosis or as playing a role in its development (Krantz & Manuck, 1984). For example, in a recent study, Krantz et al. (1999) found that among a group of cardiac patients, the patients higher in diastolic blood pressure response to stress were more likely to have cardiac events over a follow-up period of 3.5 years. Cardiovascular reactivity is measured by assessing cardiovascular changes in response to stress as compared to baseline or resting levels of those cardiovascular measures such as blood pressure, both systolic (SBP) and diastolic (DBP) and heart rate (HR). Individual differences exist where some people show large increases in response to challenging tasks (labeled “hot reactors”), while others exhibit minimal changes of cardiovascular measures. Some researchers have reviewed studies on Type A behavior and hostility, noting that such behaviors are often accompanied by heightened cardiovascular responses similar to those that are proposed to link psychosocial stress and cardiovascular disease (Contrada & Krantz, 1988).

The physiological mechanisms that might link cardiovascular reactivity and coronary artery disease are not completely clear. However, damage to the coronary endothelium may be presented as a possible explanation. Endothelial tissue damage is believed to be one of the first elements of the development of the atherosclerotic process (Ross, 1993). Stressful events result in relatively sudden changes in blood pressure and

heart rate, which are then thought to damage the endothelial lining of the vascular wall. Further, once the process of atherosclerosis has begun, these episodic reactivity events may accelerate its progression. Studies have noted a 'paradoxical vasoconstriction' in diseased coronary arteries in responses to physical and mental stress (Nabel, Selwyn, & Ganz, 1990; Yeung et al., 1991). In other words, in patients with heart disease, coronary arteries that would under normal circumstances dilate in response to increased demand actually constrict. This paradoxical vasoconstriction was found in one study to be correlated to the extent of atherosclerosis in the affected artery, and endothelium-dependent responding to an acetylcholine challenge (Yeung et al., 1991). In a more recent study of vasomotion a CAD population, using the same set of participants as those presented for the current study proposal, this paradoxical vasoconstriction was not found overall in diseased segments of coronary arteries (Kop et al., 2001). The researchers did find, however, that higher mental stress pressor responses were associated with more constriction in diseased segments of coronary arteries. Thus, mental stress resulted in significant blood pressure responses, and these blood pressure responses to mental stress were associated with vasoconstriction in diseased coronary artery segments.

Laboratory protocols to elicit physiological stress responding

Various mental stress protocols have been developed in an effort to reliably produce the acute physiological stress responding noted above in a systematic fashion. Examples of mental stress protocols include public speaking tasks, mental arithmetic (with or without harassment), anger recall tasks, and the Stroop task. This review is selective in discussing only stress protocols implemented in the proposed study. Other mental stress methods such as the star trace, the freeze frame method and protocols

eliciting anticipatory stress are not reviewed (Feldman et al., 1999; Luskin, 1999; Gregg, James, Matyas, & Thorsteinsson, 1999).

Public speaking tasks typically involve being given a topic and then being asked to give a speech for a brief period (varying across protocols, often 4 to 6 minutes) during which the participant is observed by the researcher and an audience with variations on instructions regarding content of the speech and the evaluation of the speech performance (e.g. Fichera & Andreassi, 2000; Rozanski et al., 1988). Such a task results in changes such as an increased frequency and magnitude of wall-motion abnormalities and ischemia among patients with cardiovascular disease, and in substantial changes in heart rate and blood pressure among healthy college students (Rozanski et al., 1988; Fichera & Andreassi, 2000).

Mental arithmetic is another task that has been used to elicit physiological stress responding in which the participant is asked to complete a math task, a frequent approach being to subtract 7 in series from a set of 4 digit numbers for a few minutes (with or without harassment). Carroll et al. (2000) employed the mental arithmetic protocol and elicited significant increases in heart rate and blood pressure in a large group (approximately 1650) of individuals with varying demographics (age, gender, SES). Likewise, Gottdiener et al. (1994) reported on transient wall motion abnormalities assessed by echocardiography that had been elicited by a mental arithmetic protocol. Transient wall motion abnormalities are a sign of ischemia, where the normally even and regular pattern of contraction and relaxation of the left ventricle's wall becomes disrupted and uneven. The earlier noted study on coronary vasomotion is yet another example of a study that implemented a mental arithmetic task to elicit physiological responding (Kop

et al., 2001).

Ironson and colleagues used an anger recall task (Ironson et al., 1992) in a study that assessed the effects of anger on left ventricular ejection fraction (LVEF or EF, signifying a decrease in the pump functioning of the heart). The task involved giving a speech recalling an incident that elicited anger. CAD patients participating in this task showed greater decreases in EF relative to healthy controls and that compared to two other mental stress protocols and a bicycle exercise protocol, the anger recall task led to the greatest effect in decreasing EF.

The Stroop task (Stroop, 1935) involves naming colors of each typed word in a list of words, those words being congruent or non-congruent color names. While this task is often used in cognitive psychology research such as attention and mental sets, researchers have also focused on physiological stress responding. Such research reveals heart rate and blood pressure reactivity in response to this task (e.g. Blondin & Waked, 1992). Thus there are a number of mental-stress related methods to reliably elicit cardiovascular reactivity among study participants in laboratory settings.

Associations of cardiovascular reactivity with coronary artery disease

Animal models

Cardiovascular reactivity has been linked to atherogenesis in an animal model where cynomolgus monkeys with high HR reactivity had an accelerated atherosclerotic process when compared to low HR reactors (Manuck, Kaplan, & Clarkson, 1983; Manuck, Kaplan, & Clarkson, 1985). In one of these studies, male monkeys were fed an atherogenic diet over a two year period, and then were classified as high or low HR

reactors based on their response to the "threat" of presentation of a glove, a stimulus suggesting risk of capture (Manuck et al., 1983). The high HR reactors had more extensive coronary artery atherosclerosis upon sacrifice as compared to the low HR reactors, even though they had not differed on resting HR, blood pressure, or serum lipid levels. Similar findings resulted from a study of a sample of female cynomolgus monkeys (Manuck et al., 1985). Given the design involved, however, the link between HR reactivity and the CAD process cannot be interpreted in a causal fashion (Manuck & Krantz, 1986).

Human studies

Other studies have explored the association between cardiovascular reactivity and coronary artery disease in human populations. In an early prospective study evaluating CAD development over a 23-year period in an initially healthy middle-aged male sample, blood pressure responses to a cold pressor test were predictive of subsequent CAD (Keys et al., 1971). Greater diastolic blood pressure responses to the cold pressor task were even more predictive of CAD events than two other standard risk factors that also significantly predicted CAD development, their initial systolic blood pressure and serum cholesterol levels.

In a pilot study of 13 post-myocardial infarction (MI) patients, systolic and diastolic blood pressure responses to a modified Stroop test were significantly higher among the five who had suffered a new cardiac event at follow up 39 to 64 months later (MI and/or stroke; Manuck, Olsson, Hjemdahl, & Rehnqvist, 1992). The recurrent event group did not differ from the event-free group on baseline measurements, their responses to exercise testing, fasting serum lipid and glucose levels, age, or duration of follow-up.

Krantz et al. (1991) linked greater cardiovascular reactivity with myocardial ischemia in a laboratory mental stress protocol. In a study of 39 CAD patients and 12 controls, SBP reactivity was significantly increased among the CAD patients in response to the three stressors of mental arithmetic, the Stroop task, and a public speaking task compared to controls. After dividing the CAD patients into three groups (severe, moderate, and low) based on the severity of ischemia (assessed via radionuclide ventriculography) in response to the mental stress tasks, it was shown that severely ischemic patients started out with lower double product (heart rate x SBP) levels, and reached higher double product levels during the Stroop and speech tasks. Further, SBP reactivity to stress was highest for the severely ischemic group, lowest for controls, with the mild-moderate ischemic and nonischemic patients in between, suggesting a step-wise relationship between cardiovascular reactivity and risk for ischemia. Further consideration of myocardial ischemia is contained in later sections specific to ischemia in relation to acute stress, and in relation to hostility, anger expression, and defensiveness.

In a study of unmedicated patients with stable coronary artery disease and exercise-induced ischemia, the PIMI investigators found an association between increased systemic vascular resistance during mental stress and ischemia and wall motion abnormalities (Goldberg et al., 1996). Those who developed ischemia in response to a speech task and the Stroop task had significantly greater systemic vascular resistance compared to those who did not develop ischemia in response to the two tasks.

Three older case-control studies demonstrated an association between cardiovascular reactivity and CAD (Schiffer, Hartley, Schulman, & Abelmann, 1976; Sime, Buell, & Eliot, 1980; Corse, Manuck, Cantwell, Giordani, & Matthews, 1982).

Schiffer et al (1996) examined blood pressure responses among three groups of executives while taking a quiz. The two coronary groups were one of stable angina and the other with a history of hypertension, both tapered off of medication for 72 hours prior to the laboratory study. Compared to the non-CAD control group of executives, both angina groups had higher heart rate (HR) responses. The angina with hypertension group had higher SBP and DBP responses to the quiz compared to both the angina only group and the controls.

Sime, Buell, and Eliot (1980) replicated the study using the quiz in a group of post-MI patients and healthy controls. The post-MI patients had greater DBP and lower HR responses compared to the healthy controls even with similar baseline values. Lastly, Corse et al. (1982) found that CHD patients experienced significantly greater DBP elevations during three difficult and frustrating cognitive tasks compared to non-CHD controls.

In summary, there are a number of studies implementing animal models, prospective designs and case-control designs that suggest that CVR may be a mechanism or marker for processes involved in the pathogenesis of coronary artery disease.

Acute stress and ischemia

Just as mental stress protocols have been implemented to examine cardiovascular reactivity as an indicator of possible risk for coronary artery disease, they have likewise been implemented in studies that indicate a link between acute stress and ischemia. Myocardial ischemia occurs when there is an imbalance between the supply of and demand for oxygenated blood by the heart, an event that is used clinically as a marker for CAD. While ischemia serves as a marker for CAD, an ischemic event is often “silent” in

that it occurs in the absence of pain to the individual (Kop, Gottdiener, & Krantz, 1998). Researchers thus have used this measure in the laboratory and have been able to induce myocardial ischemia in 30 to 60 percent of patients with CAD (Krantz, Kop, Santiago, & Gottdiener, 1996). While mental-stress induced ischemia can be observed reliably in the laboratory among CAD patients (e.g., Rozanski et al., 1988), it has also been observed during daily life activities using ambulatory monitoring devices and structured diaries (e.g., Gabbay et al., 1996; Gullette et al., 1997). These approaches thus help researchers observe the behaviors or specific stressful events which trigger ischemic episodes, and by extension, other cardiac events (Kop, 1999).

Ischemia assessed via electrocardiography

In noting that ischemia frequently occurs "silently," objective assessment of ischemia has been one of the tasks researchers have undertaken. Three main approaches to ischemia assessment and examples of studies using these approaches will be presented at this point. These three general approaches involve electrical activity of the heart, mechanical functioning of the heart and perfusion of the cardiac tissue (Kop, Gottdiener, & Krantz, 1998). Electrocardiographic measures of ischemia focus on shifts in the ST-segment of the electrocardiogram, with a depression of greater than 0.10 mV for more than one minute considered diagnostic of ischemia. This is representative of prolonged duration of recovery (or repolarization) in the beat-to-beat contraction of cardiac muscle. Electrocardiographic measures have been used in the laboratory and in ambulatory Holter monitoring to document how acute stress may elicit ischemia (Schiffer, Hartley, Schulman, & Abelmann, 1976; Gabbay et al., 1996).

The work of Schiffer, Hartley, Schulman, and Abelmann (1976), discussed earlier

in noting cardiovascular reactivity, provides an example of ECG assessment of ischemia. They found evidence of ischemia (greater than 1.0 mms S-T segment depression) in seven of 14 of the 'executive with angina' group in response to the audiotaped quiz, and found a positive correlation between S-T depression in response to a bicycle exercise tolerance test and to the quiz ($r= 0.63$, $p <0.01$). A second example of ECG-assessed ischemia is that of Gabbay et al. (1996), who reported on ambulatory ECG monitoring of 63 coronary artery disease patients. In comparing the continuous monitoring of the ECG to structured diaries of the patients' physical and mental activities, these researchers noted that ischemia occurred most frequently during moderately intense physical and mental activities. Adjusting for amount of time spent at differing levels of activity indicated further that the likelihood of ischemia was greatest during intense physical and stressful mental activity. The experience of intense anger was a significant trigger, and there was a corresponding significant increase in heart rate at the onset of ischemia related to anger events (1996).

Ischemia assessed by inspection of the heart's mechanical function

The mechanical functioning of the heart may provide measures of ischemia as well, in the form of transient wall motion abnormalities and transient decreases in ejection fraction (non-transient wall motion abnormalities or decreases in ejection fraction represent more severe problems such as cardiac muscle damage related to prior myocardial infarctions). Wall motion abnormalities occur when the synchronized contraction pattern of the heart becomes uneven. Ischemia is most frequently documented as wall motion abnormalities in the left ventricle, and the wall motion abnormalities occur at the specific regions where the ischemia occurs. Imaging techniques of

radionuclide ventriculography and echocardiography are employed to compare baseline measures of wall motion to wall motion during a stress task to determine inducibility of cardiac ischemia (e.g. Gottdiener et al., 1994). Ejection fraction, a ratio of blood being ejected from the ventricle compared to blood remaining in the ventricle following its contraction, is used to describe the pump function of the heart, and in exercise stress-induced ischemia studies, a less than five percent increase in ejection fraction is used as a diagnostic criterion. When cardiac demand is elevated, ejection fraction should increase, whereas in an ischemic heart, the ejection fraction doesn't increase and in some cases decreases. In mental-stress induced ischemia studies, the criteria have included a seven percent drop in ejection fraction (Ironson et al., 1992), and an eight percent decrease in ejection fraction (Becker et al., 1996; Goldberg et al., 1996).

Ironson et al., (1992) assessed left ventricular ejection fraction through radionuclide ventriculography among 18 CAD patients and nine healthy controls subjected to various stressors, both physical and mental. In the anger recall condition, seven of the 18 patients exhibited a decrease in ejection fraction of greater than or equal to seven percent, and four of the 18 patients exhibited a significant decrease in ejection fraction with bicycle exercise. The mental arithmetic and speech stressors in this study did not result in changes in ejection fraction, and there were significant differences between the treatment effects between the CAD and control groups. The Psychophysiological Investigations of Myocardial Ischemia Study investigators also implemented assessment of ejection fraction and wall motion abnormalities to determine the presence/absence of ischemia (Becker et al., 1996; Goldberg et al., 1996). They used the lack of a five percent increase in ejection fraction during bicycle exercise as a sign of

ischemia, or for mental stress, new or worsened wall motion abnormality or an ejection fraction decreased by greater than eight percent as a sign of ischemia (Becker et al.;1996; Goldberg et al., 1996). In their protocols, among 196 patients with stable coronary artery disease, 24 percent had significant decreases in ejection fraction in response to a speech task, and 25 percent had significant decreases in ejection fraction in response to a Stroop test (Goldberg et al., 1996).

Ischemia assessed by cardiac perfusion measures

Perfusion of the cardiac tissue has been assessed via positron emission tomography techniques (PET) and single-photon emission computed tomography (SPECT) and has also been linked to stress-induced changes (Deanfield et al., 1984; Giubbini et al., 1991). The unevenness of perfusion indicates the presence of ischemia, and thus provides yet a third approach to study the effects of acute stress on ischemia and by extension, coronary artery disease.

An example of a perfusion study to identify ischemia was an early study by Deanfield et al. (1984). Sixteen patients with stable angina pectoris underwent a mental arithmetic task and physical exercise. In comparing measures of perfusion with those of ECGs to detect ischemia, the authors noted that while 12 of the 16 patients exhibited perfusion deficits marking ischemia during the mental arithmetic task, only six of those were accompanied by S-T segment depression. Exercise resulted in perfusion deficits in the same ischemic segments noted during the arithmetic task, but S-T segment depression on the ECG was evident in all of these cases.

A second example of examining perfusion to assess ischemia is that of Giubbini et al. (1991). In focusing on a group of 24 patients who had recently experienced MI,

they compared results of ECG monitoring to SESTAMIBI scintigraphy measures of perfusion. While all 24 showed ECG-assessed ischemia in an exercise condition, none were found to be ischemic by ECG during a mental arithmetic task. This was contrasted to the perfusion measures, in which 20 of the 24 patients showed reversible perfusion deficits indicative of ischemic areas of the heart. These two examples and others support the contention that the more recent assessment techniques such as radionuclide ventriculography and PET imaging provide more sensitive measures of ischemia than do ECG measures of ischemia (Kop, Gottdiener, & Krantz, 1998).

Two studies noted earlier have shown a relationship between cardiovascular reactivity and ischemia (Krantz et al., 1991; Golberg et al., 1996). Many cardiovascular reactivity studies and ischemia studies have utilized similar mental-stress inducing protocols as described earlier, with some of the studies assessing both reactivity and ischemia. It may consequently be argued that the underlying mechanisms are similar as well in linking reactivity and ischemia to coronary artery disease and its major events. This concept will be touched upon later in the introduction in consideration of theoretical models and mechanisms relating anger, hostility, and defensiveness to coronary artery disease.

Section summary: Acute stress and cardiovascular disease

In summary of this section of the introduction regarding acute stress and cardiovascular disease, acute stress has been shown to elicit ischemia in a number of studies. Likewise, cardiovascular reactivity has been associated with the onset of ischemia among cardiovascular disease patients, as well as related to coronary artery disease using other CAD endpoints in both cross-sectional and longitudinal studies.

Assessment approaches for cardiovascular reactivity and ischemia have been reviewed, with brief consideration given also to examples of studies implementing such measures.

Behavior, personality, and CAD: Anger, hostility, and defensiveness

The following section will address specific behavioral and personality characteristics that have been studied in relation to coronary artery disease, those of anger, hostility and defensiveness. There will be an initial discussion of personality definitions and assumptions, followed by a review of selected studies regarding the relationship between anger, hostility, defensiveness and cardiovascular reactivity in acute stress situations. Next, the relationship between these same behavioral and personality characteristics will be considered as pertaining to their potential role in the development of ischemia in acute stress situations. Lastly, theoretical models and mechanisms relating anger, hostility, and defensiveness to CAD will be considered.

Personality Traits: Definitions and Assumptions

Personality has been defined by Carver and Scheier (1996) as a dynamic organization within a person comprised of psychophysical systems creating the person's characteristic patterns of behavior, thoughts, and feelings. Operating under that definition, the behaviors, thoughts, and feelings provide an indication of the underlying psychophysical systems, a window into that organization suggesting the composition of the individual's personality. Another conceptualization of personality is the dispositional perspective, which suggests that a person's dispositions may be defined as relatively stable and enduring qualities which are unique to that individual, and that personality

consists of a number of different dispositions possessed by the individual (Carver and Scheier, 1996). These dispositions may be interpreted as traits, which differ in individuals in the amount that each characteristic may be evident in their personality. Traits refer to characteristics or dispositions on which people differ in dimensions or degree, so that a personality trait may be quantified as a continuous variable, such that the degree of presence versus absence of the characteristic is distributed across a population.

Consequently, many attempts have been made to characterize personality traits via descriptions of behaviors, thought, and feelings. Most of these research efforts rely upon self-report measures of various characteristics (e.g. Cattell, Eber, & Tatsuoka, 1977- the 16PF; Eysenck & Eysenck, 1985- the EPQ; Digman, 1990- reviews the "Big Five"). Such self-report measures are seen as indicative of certain patterns of behavior, and health psychologists as well as personality psychologists have extended the certain patterns of behavior to being seen as predictive of types of physiological responding, such as the field of cardiovascular reactivity being linked to personality traits (e.g. Fichera & Andreassi, 2000; Schwebel & Suls, 1999).

Trait approaches to personality view basic differences between individuals as being quantitative rather than qualitative, and assessments thus involve specifying the amounts of various characteristics (Carver & Scheier, 1996). Such approaches also presume that traits are enduring characteristics that also may be quantified, expecting that such characteristics are consistent across time and situation. Thus in looking at patterns of behavior, thoughts, and feelings, possessing more of a certain personality trait suggests that one would see more of the behaviors, thoughts, and feelings characteristic of that personality trait (Carver & Scheier, 1996). "Seeing more" of the behaviors, thoughts, and

feelings would suggest higher frequencies of the behaviors, thoughts, and feelings, or longer duration of associated states (to include emotion). One of the personality traits of particular interest in the field of health psychology has been that of hostility, and its relationship to the emotion of anger as well as aggressive behaviors.

While trait approaches continue to be studied in personality research, there have been criticisms to the approach. Mischel (1968) challenged the idea that self-report measures represented traits, or stable aspects of personality across a wide range of settings. He pointed to relatively modest correlations between self-reports and behavior, ranging from .20 to .30, which he termed personality coefficients. He argues that behavior is highly situation specific, which would seem to contradict the concept of dispositions or traits being manifest across situations and settings. This seeming disconnect between self-reports and behavior serves as a component of the rationale for testing relationships between self-reports and physiological responding to be discussed later.

Anger, hostility and defensiveness/repression- definition and assessment

Definitions

The general conceptualizations of anger suggest it may be viewed as an affect or state, whereas hostility may be interpreted as an attitude or trait, and aggression represents behaviors derived from hostility or anger (Spielberger, Reheiser, & Sydeman, 1995; Smith, 1994; Barefoot & Lipkus, 1994). Anger is a multidimensional construct frequently characterized as an emotional state that varies in intensity. However, different definitions emphasize different aspects, such as the cognitive, behavioral, or

physiological dimensions of the experience of anger. For example, Kaufman (1970) defined anger as "...an emotion that involves a physiological arousal state coexisting with fantasized or intended acts culminating in harmful effects on another person" (p. 12). In contrast, Berkowitz (1993) suggested that anger might not have a particular goal, but that it may lead to aggressive motor behavior that does have a deliberate intent to harm, hurt or injure another person or object. Finally, Averill (1983) presents ''anger displays" as socially defined behaviors learned via reinforcement history.

Kassinove and Sukhodolsky (1995) have differentiated forms of aggression as being either hostile (motivated by anger) or instrumental (goal directed, not motivated by anger). Similarly, another conceptualization of anger and hostility has been characterized by what Spielberger and colleagues (Spielberger, Jacobs, Russell, & Crane, 1983) have termed the AHA! Syndrome (anger, hostility, & aggression):

> Anger usually refers to an emotional state that consists of feelings that vary in intensity, from mild irritation or annoyance to intense fury and rage. Although hostility usually involves angry feelings, this concept has the connotation of a complex set of attitudes that motivate aggressive behaviors directed toward destroying objects or injuring other people.. .While anger and hostility refer to feelings and attitudes, the concept of aggression generally implies destructive or punitive behavior directed towards other persons or objects. (p.16)

Spielberger and colleagues (Spielberger, Reheiser, & Sydeman, 1995) note that anger serves as the core of the AHA! Syndrome. However, further distinctions between hostility and aggression are often ignored, as aggression and hostility have been used almost interchangeably in the research literature (Spielberger et al., 1995).

Physiological correlates of anger and hostility

While the above noted researchers have focused on the feelings and attitudes

associated with anger and aggression, the physiological component of the anger experience is critical to discussion of the relationship of hostility and anger expression to cardiovascular reactivity and disease (e.g. Williams, Barefoot, & Shekelle, 1985). The specific physiological changes that occur during anger experiences include higher autonomic arousal evidenced by elevated systolic and diastolic blood pressure, and heart rate, as well as by muscle tension (often in the face and hands), facial flushing, and a sensation of increased body temperature related to adrenal and other endocrine changes (Williams, Barefoot, & Shekelle, 1985; Kassinove & Sukhodolsky, 1995).

Anger, hostility, and health

Cardiovascular risks have also been related to hostility. In a review of longitudinal studies relating anger and hostility to CAD, Rozanski and colleagues noted mixed results among eleven studies of initially healthy subjects, noting the studies varied in quality and length of follow-up (Rozanski, Blumenthal, & Kaplan, 1999). Their review of longitudinal studies of CAD patients relating hostility and anger to recurrent events noted four studies all indicating an increase in relative risk associated with anger and hostility. Further, they noted findings that CAD patients with high levels of hostility have a greater rate of restenosis after angioplasty (Goodman, Quigley, Moran, Meilman, & Sherman, 1996) and exhibit more rapid progression of atherosclerosis assessed by serial carotid ultrasonography over time (Matsumoto et al., 1993; Julkunen, Salonen, Kaplan, Chesney, & Salonen, 1994).

The interest in the possible role of hostility in CAD is reflected in part by the early work of Rosenman and Friedman on the Type A Behavior Pattern (Friedman & Rosenman, 1959; 1974; Rosenman and Friedman, 1974). These cardiologists noted a

distinct constellation of behaviors in many of their patients that they labeled Type A coronary-prone behavior because they considered these behaviors risk factors for coronary heart disease (CHD). Type A behaviors included competitiveness, impatience, a sense of time pressure, and aggressive and hostile behavior. The patients who did not display the Type A behavior pattern were identified as Type B (absence of the Type A behaviors). A structured interview has been used to assess the presence or absence of Type A behaviors overall (Rosenman et al., 1975), but many researchers have evaluated separate ratings of each of the behavioral components of the Type A behavior pattern, including specific ratings of hostility (e.g., Matthews, Glass, Rosenman, & Bortner, 1977). However, it is important to note that Barefoot and Lipkus (1994), in a review of measurement issues regarding anger and hostility related to health, found that the correlations between Structured Interview hostility ratings and self-report measures are typically low, with no clear information regarding convergent or discriminant validity of interview-based assessments. These results, in combination with considerations such as that of Mischel (1968) regarding the seeming lack of correlation between self-report and behavior may explain why self-report measures of anger and hostility in health outcome studies have yielded conflicting results (Hemingway & Marmot, 1999; Miller, Smith, Turner, Guijarro, & Hallet, 1996).

Suppression, repression, and defensiveness and health

Perhaps another explanation for conflicting results among self-report measures of anger and hostility in health outcome studies is the influence of yet other characteristics related to anger and hostility: suppression, repression and defensiveness (Siegman, 1994; Mann & James, 1998). Suppression of an emotion is conceptualized as being aware of an

emotion but simply not expressing it, a conscious holding in of the emotion, whereas repression may be interpreted as a denial of the emotion without awareness (an nonconscious or subconscious effort; Siegman, 1994). Suppressed anger and hostility have been presented as predisposing characteristics of essential hypertension (Dunbar, 1943; Johnson, Gentry, & Julius, 1992). More recent reviews relating suppression or repression to essential hypertension suggest that overall results are inconclusive (Jorgensen, Johnson, Kolodziej, & Greer, 1996; Suls, Wan, & Costa, 1995), but further investigations into repression and suppression have linked them to impaired immune function (i.e. Pennebaker, Kiecolt-Glaser, & Glaser, 1988) and cancer (i.e. Jensen, 1987).

Defensiveness, as a related construct, has also received attention as a possible psychosocial factor in various health problems. Defensiveness may be defined as the conscious or unconscious tendency not to report socially undesirable aspects of oneself, the concern with a positive self-representation, and the need to be viewed in a positive manner by others (Crowne & Marlowe, 1964; Paulhus, 1984). Thus a defensive individual would be expected to more frequently repress or suppress negative emotions for purposes of positive self-representation. Mann and James (1998) reported that defensiveness was associated with essential hypertension, with a relative risk of 3.63 after statistically controlling for age, gender, and Body Mass Index when comparing 50 normotensive study participants to 74 hypertensive participants. Even more recently, Rutledge and Linden (2000) presented longitudinal data supporting a defensiveness/essential hypertension relationship. After a three year follow-up, 127 initially normotensive males and females were found to have a differential risk of hypertension development, in that twenty percent of the highly defensive participants had

developed hypertension, whereas only 4.5 percent of participants low in defensiveness had developed hypertension. Membership in the highly defensive group was thus associated with more than a seven-fold risk for developing hypertension over the three-year period, after statistically adjusting for general risk factors (including smoking, exercise levels, alcohol consumption and body fat composition). Thus repression/suppression and defensiveness have been presented as psychosocial factors that influence health outcomes. Their potential relationships as mediating factors for anger/hostility influencing cardiovascular reactivity and ischemia will be discussed later.

Hostility assessment

As for the assessment of hostility, the two most common measures involve the Type A Structured Interview (SI), and the Cook-Medley Hostility (Ho) Scale derived from the Minnesota Multiphasic Personality Inventory (Cook & Medley, 1954). The Structured Interview is an interpersonal interview in which the interviewer challenges and interrupts the subject in order to elicit a behavioral response (Friedman & Rosenman, 1974). The interview can be scored regarding both content and behavioral responses of hostility and other components to include response latency, vigorous speech stylistics, anger suppression, and competition for control of the interview, all considered elements of the Type A behavior pattern. An individual rated as high in SI-derived hostility is described as argumentative and challenging in response to interviewer questions, his voice indicating boredom and surliness, and his responses to specific questions may be characterized by impatience, anger, and irritability (Dembroski, 1983). Thus the interview yields a score reflecting judgment of overt behavioral potential for hostility.

The Cook-Medley Hostility Inventory (Cook & Medley, 1954) is the most

frequently used self-report measure of hostility. This hostility scale was initially developed to differentiate between teachers having good versus bad rapport in their relationships with students, but has since become popular among health outcomes researchers as evidence accumulates suggesting it is an important marker for health status (Smith, 1992).

Several factor analyses have described varying components of hostility within the overall measure. Williams and colleagues presented a factor labeled mistrust (in people's good intentions; 1985). Costa, Zonderman, McCrae, and Williams presented a component labeled paranoid alienation (1986). Blumenthal, Barefoot, Burg, and Williams reported an anger component (1987). Some researchers (e.g. Rosenman, Swan, & Carmelli, 1988) have suggested the hostility scale is most appropriately deemed a measure of psychopathology rather than hostility. Thus, findings from factor analyses have resulted in inconsistencies, with different analyses yielding one factor (e.g. Greenglass & Julkunen, 1989), two factors (e.g. Costa, Zonderman, McCrae, & Williams, 1986), and three factor solutions (e.g. Lipkus, Barefoot, Beckham, & Haney, 1993).

A last example of how the Cook-Medley Hostility Scale has been interpreted is the work of Barefoot, Dodge, Peterson, Dahlstrom, and Williams (1989). They conducted an analysis of six subsets (classified a priori) of the Cook-Medley hostility scale, from which a Composite Hostility (Ho) score best predicted survival among a set of 118 initially healthy law students over a 29-year follow-up. The survival analysis was done for total mortality and a separate analysis was not conducted for CAD mortality because only 6 of the 13 total deaths were attributed to CAD. The Composite Hostility score was a combination of three subscales of the Cook-Medley Hostility Scale (cynicism, hostile

affect, and aggressive responding). In general, researchers suggest the Ho scale reflects cynical beliefs and mistrust (Barefoot & Lipkus, 1994). Other studies have continued to implement the Composite Hostility score in assessing hostility-health relationships (Suarez & Williams, 1989; Helmers et al., 1993; Helmers et al., 1995).

Overall, the relationship between the two measures of Cook-Medley Hostility and the SI-derived Potential-for-Hostility has been found to be moderate. For example, in 131 patients referred for diagnostic cardiac catheterization, the correlation between the two measures was 0.37 (Dembroski, MacDougall, Williams, Haney, & Blumenthal, 1985). The moderate correlation suggests that while there is some overlap between the scales they are not identical.

Further, Kassinove and Sukhodolsky (1995), in noting the multiple measures for (and inconsistent use of terms for) hostility and anger, have highlighted the differential representations, not just in the nature of the measurements, but also of the constructs of hostility and anger that are present in the research literature. For example, the different perspectives on the anger construct may emphasize separate aspects of the response dimensions (e.g. verbal or non-verbal, directed or not directed toward the anger-inducing person/event), temporality (i.e. state vs. trait), and generality of response. Similarly, use of such constructs may differ in reference to the stimuli that elicit angry, aggressive, or hostile responses, such as internal, external, social, clear or ambiguous stimuli, or stimuli differing in magnitude.

Assessment of anger expression, repression, defensiveness

One of the parameters in which anger and hostility measures have varied is the

issue of repression or defensiveness. The tendency towards suppression has been characterized as “anger-in” and “anger-control” by Spielberger in his Anger Expression Scales (also characterized as low “anger-out”, Speilberger et al., 1985). Both anger-in and anger-control are obtained by self-report with ten items for each factor regarding the likelihood of the individual to direct the anger in without expression, or to control the anger experience (Spielberger et al., 1985). Such measures, however, have been criticized as failing to differentiate between suppressing (simply not expressing) anger and repressing anger (Siegman, 1994), and the relationship between these anger expression measures and defensiveness is not entirely clear.

An example of anger expression measures related to cardiovascular reactivity is the work of Faber and Burns (1996), who noted that in a sample of 32 undergraduate women, those who had scored lower on anger-out showed sustained elevated systolic blood pressure responses following interpersonal harassment. While some researchers make distinctions on whether the lack of expression of the anger and hostility is done with awareness or consciousness (suppression, as opposed to the unconscious repression), others tend to discuss the concept without making this distinction, as pointed out by Siegman (1994). For example, Brosschot and Thayer draw upon examples of both repression and suppression in proposing their model of the association between hostility and cardiovascular disease, referring to the concept as ‘anger inhibition’ (1998). Others have also implemented anger suppression measures in their research (with the SI, coding anger suppression, such as in Anderson & Lawler, 1995 and with Framingham Anger-In, as in Haynes, Feinleib, & Kannel, 1980). Anger expression measures generally are not equated to defensiveness, as defensiveness encompasses more than just suppression of

anger. In other words, suppression of anger expression may be one of many components in a defensive personality style.

Defensiveness has been measured by the Marlowe-Crowne Scale of Social Desirability (MC SDS; Crowne & Marlowe, 1960; as in Weinberger, Schwartz, & Davidson, 1979). Crowne and Marlowe described their scale as measuring inhibition, "defensiveness and protection of self-esteem" (1964, page 206). The Marlowe-Crowne SDS was developed to contrast with the Edwards Social Desirability Scale (Edwards, 1954), which was developed entirely from Minnesota Multiphasic Personality Inventory items (MMPI; Hathaway & McKinley, 1943). The MC SDS contains one exact and four approximate replications of *L* items, and one repetition of a *K* scale item (*L* and *K* scales from the MMPI) among its 33 items. There is, however, a .40 correlation between the MC SDS and the *K* scale, and a .54 correlation between the MC SDS and the *L* scale. Crowne and Marlowe indicated one of their goals was to separate out psychopathology content from social desirability content in their items, indicating that both the MMPI and the Edwards SDS assess social desirability confounded with psychopathology.

In Weinberger et al.'s study of anxiety, participants scoring high on the Marlowe-Crowne scale were termed repressors (low anxious/high Marlowe-Crowne score) or defensive high anxious (high anxious/high Marlowe-Crowne score; 1979). As Weinberger and colleagues have paired anxiety and defensiveness, other researchers have focused on the relationship between hostility and defensiveness, as in the work of Jamner and colleagues, who found greater cardiovascular reactivity in a sample of 33 healthy males among those participants who were high in both hostility and defensiveness (Jamner, Shapiro, Goldstein, & Hug, 1991).

More recently, studies have paired Spielberger' s anger-out subscale with the Marlowe-Crowne Social Desirability Scale (Burns, Evon, & Strain-Saloum, 1999; al'Absi, Bongard, & Lovallo, 2000). Burns, Evon, and Strain-Saloum referred to participants who scored high on both scales as defensive anger expressors, and referred to participants high on the MarloweCrowne only as anger repressors (1999). They noted that anger repressors tended to report lower levels of anger, yet evidenced cardiovascular reactivity similar to that of high anger expressors. Al'Absi, Bongard, and Lovallo reported greater adrenocortical stress responses in a group of participants high in Anger-Out and defensiveness subjected to tasks of mental arithmetic and a public speaking task related to personally sensitive issues (2000). Their finding of greater adrenocorticotropin hormone levels among high Anger-Out/high defensive participants was explained as an indication of emotional distress activating the HPA axis (hypothalamus- pituitary-adrenal axis). This emotional distress was hypothesized to be greater among the high Anger-Out/high defensive participants due to their conflict between an outward anger expression style and concerns about social evaluation (defensiveness). In their discussions both groups suggest an interactive effect of style of anger expression with a general tendency towards defensiveness in the elicitation of cardiovascular reactivity, even though one focused on significant results related to the low Anger-Out/high defensiveness group (Burns et al., 1999) whereas the other focused on significant cardiovascular reactivity and neurohormonal activity among participants scoring high in both measures. Thus, while measures of anger-in, anger-control, and other measures have focused on anger inhibition, there is a degree of conceptual overlap between anger expression measures and defensiveness, as defensiveness may affect the likelihood of

anger expression.

Anger, Hostility, Defensiveness/Repression and Cardiovascular Reactivity

Anger and hostility have been postulated to play a role in cardiovascular reactivity in a large number of studies, both among healthy individuals and among cardiovascular disease patients. The results overall for such studies, however, are inconsistent, with some studies not finding relationships between anger, hostility, and cardiovascular reactivity, and by extension, coronary artery disease (Miller et al., 1996; Hemingway & Marmot, 1999; Rozanski, Blumenthal, & Kaplan, 1999). Given such conflicting results, some researchers have focused on the possible impact that repression or defensiveness might have on the hostility/CAD relationship (i.e. Miller, 1993; Burns, Evon, & Strain-Saloum, 1999). This section of the introduction will review a number of studies inspecting the role of the psychosocial variables of anger and hostility, on cardiovascular reactivity, and also the role of defensiveness or repression on cardiovascular reactivity.

Anger, hostility, and cardiovascular reactivity

Many researchers have assessed the relationships between anger and hostility and cardiovascular reactivity. One example of anger being related to cardiovascular reactivity (CVR) is that of the research of Anderson and Lawler (1995). In this study, one of few to study women exclusively, the anger recall interview was used to elicit cardiovascular responses among 58 women classified as Type A or Type B by Structured Interview. The researchers reported that Type A women had greater systolic reactivity when their anger was directed towards a subject labeled a "frustration of autonomy needs." They further

reported that suppression of anger led to greater pressor responses than did assertive anger expression. This study and most of the others to be reviewed note that the experience and expression of anger appears highly situation-specific, in that those rating high in trait anger or hostility will only be shown to have greater cardiovascular reactivity under stressful or anger-provoking settings, most often manipulated and measured in the laboratory setting.

In another example of hostility associated with CVR, Bongard, al'Absi, and Lovallo (1998) reported on an interaction between Cook-Medley hostility and anger expression in eliciting cardiovascular reactivity in a sample of young men participating in tasks of mental arithmetic and public speaking. Participants that were high in Anger-Out and low in Cook-Medley hostility displayed the greatest increases in heart rate and blood pressure. This group displayed what they referred to as a "mismatch" between the participants' hostile cognitions and habitual anger expression style, in that their behaviors reflect anger, whereas their cognitions are not high in hostility (1998). Thus, in the High Anger-Out/Low Hostility group, this mismatch was presented as a possible explanation for the group's increased cardiovascular reactivity. This "mismatch" hypothesis is in contrast to studies to be reviewed in following sections which suggest a combination of high defensiveness and high hostility lead to greater cardiovascular reactivity (e.g. Jamner, Shapiro, Goldstein, & Hug, 1991; Helmers et al., 1995; Helmers & Krantz, 1996).

Benight et al. (1997) presented findings of heightened cardiovascular reactivity in response to two mental stress tasks among six men with CAD and nine healthy male controls. Both an anger recall task and an anger with helplessness speech task elicited

increased heart rate and blood pressure among patients with CAD and among the healthy controls as well. While both normals and CAD patients reported significantly higher anger following the two tasks, no analysis was presented regarding differing degrees of cardiovascular reactivity as a function of anger ratings within the task conditions. This prevents one from interpreting their findings as revealing that relatively greater anger results in higher levels of cardiovascular reactivity. Benight and colleagues also assessed myocardial perfusion, which will be addressed in a later section relating anger and hostility to myocardial ischemia (1997).

Suarez and Williams reported findings relating hostility to cardiovascular reactivity (CVR) in college males in response to a mentally stressful task (Suarez & Williams, 1989; 1990). Those that scored higher on the Cook-Medley hostility scale had greater increases in diastolic blood pressure (DBP) and longer recovery times for systolic blood pressure (SBP). Further, during the anagrams task with harassment, the high hostile group reported greater intensities of anger, irritation, and tension, whereas the high and low hostile groups did not differ on tasks not involving harassment. Anger ratings were positively correlated to blood pressure reactivity in the high hostile group, but not in the low hostile group, suggesting a mediating role of anger in the hostility-cardiovascular reactivity relationship for this group (1989). In combining six different anger and hostility measures, the researchers presented a factor analysis yielding two factors, labeled antagonistic hostility and neurotic hostility (1990). The male college students scoring high in antagonistic hostility had significantly greater SBP responses during anagrams with harassment, and slower SBP recovery following the task. The researchers also analyzed their data using the Composite Hostility score (Cook-Medley subscales of

Hostile Affect, Cynicism, and Aggressive Responding; Barefoot et al., 1989), finding the Composite Hostility scale to be a better predictor than the full Cook-Medley for cardiovascular reactivity (1989). These studies again suggest the necessity of an anger-provoking situation for the hostility-cardiovascular reactivity relationship to be revealed. Such studies support the work of Suls and Wan (1993), who presented a meta-analysis of studies on the relationship between hostility and cardiovascular reactivity, suggesting that social stress as a provocation was essential for manifestation of the hostility/CVR relationship.

Anger, hostility, and cardiovascular reactivity- negative findings

A number of other studies have also shown a positive relationship between hostility scores and cardiovascular reactivity in laboratory settings with mental stress manipulations (Weidner, Friend, Ficarrotto, & Mendell, 1989; Hardy & Smith, 1988; Smith & Allred, 1989, Houston, Smith, & Cates, 1989). There are also studies, however, reporting no relationship between hostility and cardiovascular reactivity, with varying explanations (Smith & Houston, 1987; Sallis, Johnson, Trevorrow, Kaplan, & Hovell, 1987; Anderson, Williams, Lane, & Monou, 1984). Smith and Houston explored cardiovascular reactivity in 60 male undergraduates undergoing the Stroop Color-Word task and mental arithmetic with harassment, yet found no significant relationship between reactivity and Cook-Medley hostility (1987). They further found no relationship between reactivity and anger expression scales (Anger-In, -Out, and -Discuss) from the Framingham Heart Study, but found a negative correlation between hostility and the Anger-Out scale. Given that neither hostility nor anger expression measures were significantly associated with cardiovascular reactivity in this group and the negative

correlation between hostility and Anger-Out, the authors suggested that the relation between hostility or anger expression and coronary heart disease may be mediated or moderated by the other variable. The possibility that the effects of hostility on cardiovascular disease may be moderated by anger or its expression will be an important element of the study hypotheses to be discussed later.

Sallis and colleagues (1987) reported no significant relationship between Cook-Medley hostility and cardiovascular reactivity in response to a cold pressor task and a mental arithmetic task. Among the 46 males and 30 female participants (mean age of 36), there were also no gender by hostility interactions. The one major difference between this study and others noted to this point is that the mental arithmetic task was implemented without harassment. Other studies have noted the importance of an interaction between hostility and a significant situational stressor, such as an anger-provoking manipulation, suggesting that the nonsignificant findings in this study may be attributable to the lack of harassment during the task.

The failure to find a significant relationship between hostility and cardiovascular reactivity in another study is not attributable to a failure to harass the participants (Anderson et al., 1984). In this study, male college students performed a Word Identification Task while being criticized by a confederate. The task with harassment resulted in significant cardiovascular reactivity, but these responses were not differentiated by levels of hostility. As the study was presented in abstract form, without number of subjects reported, it is quite possible that the study lacked sufficient power to demonstrate the hypothesized relationship.

In one study, researchers compared state and trait anger measures as related to

ambulatory blood pressure measures among 100 college students (42 men, 58 women; Porter, Stone, & Schwartz, 1999). For seven days, the students completed state anger measures in response to anger-provoking situations, having already completed trait anger measures. Porter and colleagues noted only moderate correlations between state and trait anger measures over a seven-day period, and also that neither trait nor state anger expression scales were related to blood pressure levels. They found no differences between genders regarding anger measures, with slightly higher SBP and DBP levels among males.

While this study is notable for examining these relationships outside of the laboratory, the failure to find a relationship between anger expression and blood pressure levels might be attributable to the methodology and analysis implemented. Blood pressure was monitored on only one day, and aggregated, and anger expression scores were also aggregated, preventing comparison of specific anger events to blood pressure and heart rate reactivity during those specific times. Further, participants reported an average of only 0.87 (SD 1.4) incidents per day on those monitored days. This study does provide some support for Mischel and others' arguments about the seeming lack of accuracy for personality measures in predicting emotional states and behavior, however, as correlations between state and trait anger expression measures were moderate (0.37). These moderate correlations suggest other factors were influencing state anger. Further, situational variables such as the target of the anger, the presence or absence of that target, and whether the incident was in public or private also significantly predicted state anger expression. These situational requirements buttress the argument for settings and circumstances influencing reactivity as opposed to having reactivity being simply a

manifestation of underlying personality characteristics alone.

Among the studies presented with no relationship found between hostility and anger and cardiovascular reactivity, explanations have included the possible lack of power in numbers and also strength of manipulation, in that a sufficiently anger-producing event is needed to reveal differential cardiovascular reactivity. The other potential explanation for failure to find such a relationship was that anger or hostility might be mediating the effects of each other, requiring certain levels of both factors or personality characteristics to lead to cardiovascular reactivity. Yet another possible explanation that has been presented in general terms to this point is that the inhibition, suppression, or repression of the anger or hostility may also mediate the relationship.

Anger inhibition and defensiveness associated with cardiovascular reactivity

Inhibition of anger has been studied as noted earlier as Anger-In by the Framingham study, Spielberger' s Anger Expression scale, and SI-derived ratings of Anger-In (Haynes, Levine, Scotch, Feinleib, & Kannel, 1978; Spielberger et al., 1985; Dembroski, MacDougall, Williams, Haney, & Blumenthal, 1985; Dembroski, 1983). Also, hostility and anger measures have been paired with or compared against measures of defensiveness and social desirability using the Marlowe-Crowne Social Desirability Scale (Crowne & Marlowe, 1964). Cardiovascular reactivity studies using such measures have met with mixed results. While a number of studies have noted significant relationships between inhibition of anger or defensiveness and hostility and CVR (e.g. Helmers & Krantz, 1996; Jamner, Shapiro, Goldstein, & Hug, 1991; Burns, Evon, & Strain-Saloum, 1999), others have not (Mills, Schneider, & Dimsdale, 1989; Smith & Houston, 1987; Powch & Houston, 1996).

Helmers and Krantz (1996) reported gender differences among healthy individuals in response to math and speech tasks such that defensive hostility led to greater systolic blood pressure responses in men, but was not related to blood pressure responding in women to the same tasks. A mental arithmetic without harassment task and a speech task (about personal faults and problems) were given to 33 men and 34 women. Participants were classified by levels of Cook-Medley hostility (Ho; high or low with dichotomous split) and by levels of defensiveness with the Marlowe-Crowne Social Desirability Scale (MC; high or low with dichotomous split). This resulted in four groups, labeled a) Defensive Hostile (DH- high Ho, low MC), b) Low Hostile (LH- low Ho and MC), c) High Hostile (HH-high Ho, low MC), and d) Defensive (Def- low Ho, high MC). While the DH men exhibited greater SBP levels than LH, Def, and HH men during the tasks, they were also higher at baseline measures, with a marginal significance for a Hostility X Defensiveness interaction [$F(1, 29) = 3.7$, $p = .06$]. Women did not exhibit these differences. In noting a significant Hostility X Defensiveness interaction, DH women had only intermediate SBP levels, with the Defensive and Low Hostile groups having greater responding. There were trends suggesting that Low Hostile and Defensive Hostile men demonstrated greater diastolic blood pressure responding, which was not found in the women. Heart rate changes were associated with tasks, but not with hostility X defensiveness interactions. This study then, presents evidence suggesting that among healthy individuals, the interaction of hostility and defensiveness may provide significant associations with blood pressure levels in studies that previously did not find associations using hostility alone, and that this may apply especially among men. It is further possible that the differences during the mental arithmetic task could have been

greater in this study by adding harassment as a component of the manipulation.

In conceptualizing defensiveness as the tendency not to report socially undesirable aspects of oneself, one may argue that this tendency is in opposition to the hostile attitudes and cynical outlook as represented by high Cook-Medley hostility scores. In fact, Jamner, Shapiro, Goldstein, and Hug (1991) suggest that high defensiveness as assessed by the Marlowe-Crowne Social Desirability Scale indicates a tendency to deny or inhibit anger. Janmer and colleagues reported increased cardiovascular reactivity in ambulatory blood pressure and heart rate among those scoring high in both hostility and defensiveness in a field study of 33 healthy male paramedics (1991). The paramedics were monitored over a 24-hour work shift, and completed diary entries about their activities coincident with blood pressure readings. Using similar classifications as those explained above (Helmers & Krantz, 1996), these authors noted the negative association between the measures of hostility and defensiveness, as only six individuals were classified as High Defensive/High Hostile, and only six were Low Defensive/Low Hostile. There were ten classified as Low Defensive/High Hostile and eleven classified High Defensive/Low Hostile. Among the various work settings that were found to interact with Defensive Hostility, the authors pointed to significant heart rate increases among this group in hospital settings involving interpersonal conflict. Their explanation of the findings was that rating high in both characteristics leads to the effect of rendering individuals more conflicted or ambivalent about inhibiting expressions of anger and hostility— they experience the negative emotions more frequently, and thus must work even harder at efforts to deny or inhibit the expression of anger and hostility (Jamner et al., 1991).

Others suggest that this denial or inhibition of anger occurs at an unconscious level, a pattern of repression rather than a conscious suppression. As noted earlier, Burns, Evon, and Strain-Saloum (1999) reported that anger repressors (high Marlowe-Crowne Social Desirability scores combined with low Spielberger Anger-Out) under interpersonal harassment exhibited higher cardiovascular reactivity but reported lower anger levels. The classifications for the groups were low anger expressor, anger repressor, high anger expressor, and defensive anger expressor based on median splits of the two scales in a similar manner as the two previous studies relating defensiveness and hostility. The study involved 102 college students (51 male) who underwent one of two conditions, harassment or non-harassment, in which they told stories when shown cards from the Thematic Apperception Test (TAT; Murray, 1943). Within the harassment condition, there were 4 cards without harassment to establish a baseline. While significant differences among groups classified by MC and Spielberger Anger-Out existed with anger repressors showing heart rate reactivity similar to high anger expressors, they reported lower anger levels than did the high anger expressor group. Anger repressors did not differ significantly from the other groups in terms of systolic and diastolic blood pressure. The researchers did not present analyses of gender effects.

The researchers presented the anger repressors as being individuals who experience anger physiologically and behaviorally, yet deny their anger in a form of self-deception (Burns et al., 1999). Burns and colleagues interpret this as a distinct anger management style that results in a discrepancy between cardiovascular reactivity and acknowledged anger. This study, like others, notes the importance of anger or stress-provoking situations (e.g. Burns, 1995; Burns & Katkin, 1993; Suarez & Williams, 1990)

to elicit the differential CVR responding, highlighting that repression effects are most apparent when social evaluative cues are pronounced (e.g. Newton & Contrada, 1992). This study differs from the previous two studies in focusing on the pairing of low anger expression and high social desirability, whereas the two studies of defensiveness and hostility focused on individuals rating high in both social desirability and anger expression. The authors provide no clear explanation for one approach versus the other in best explaining the interaction between the two factors. Across the three studies there is also the difference that Helmers and Krantz (1996) noted differences in pressor responses whereas the other two found their differences in heart rate reactivity (Jamner et al., 1991; Burns et al., 1999). Siegman (1994) has argued that expressed anger may be associated primarily with pressor responses, whereas repressed anger may be linked to heart rate reactivity, however the results across these three studies noted as well as others are inconsistent on this point.

Al'Absi, Bongard and Lovallo (2000) use the term "mismatch" as opposed to "discrepancy" as they note elevated adrenocorticotropin responses (signifying heightened hypothalamic-pituitary-adrenocortical, or HPA, responding) among young healthy males participating in mental arithmetic and public-speaking tasks. Participants high in both anger-out (Spielberger's Anger Expression Inventory) and defensiveness (Marlowe-Crowne Social Desirability) exhibited heightened HPA and cardiovascular reactivity. The stress responding to the socially salient mental stress tasks was accentuated by the mismatch between the preferred anger expression style and the desire to present oneself in a positive manner. More consideration of the role of defensiveness and repression or inhibition of hostile and angry responding and possible mechanisms relating those

psychosocial variables to cardiovascular responding and disease is provided toward the conclusion of the next section of the introduction.

Anger inhibition, defensiveness, and cardiovascular reactivity- negative findings

Not all studies of the role of anger inhibition or defensiveness suggest a significant relationship with cardiovascular reactivity, however (Mente & Helmers, 1999; Shapiro, Goldstein, & Jamner, 1995). Mente and Helmers inspected defensive hostility among a total of 46 young men (aged 18-45 years) with extreme scores on the Cook-Medley Hostility and the Marlowe-Crowne Social Desirability scales (1999). The participants were divided into four groups, with extreme high and low scores of the two scales as the parameters. The participants completed a cold pressor task, a public speech task, and an anger recall task. Defensive Hostile participants (high MC and high Ho) and Nonhostile nondefensive (low MC and low Ho) individuals displayed increased diastolic blood pressure reactivity to the cold pressor task, but there were no significant differences in reactivity between groups during the two mental stressors. In discussing their results in relation to other studies in the area, they suggest that their laboratory stressors may not have been sufficiently stressful to elicit the changes noted in other studies, in which researchers assessed stressful situations in daily life, or where participants were provoked by the experimenter. Also of note is the approach of selecting for extreme scores for the hostility and defensiveness measures. This selection for extreme scores would be expected to highlight the effects of defensive hostility, but results were nonetheless insignificant in response to the mental stress tasks.

The other study reporting no relationship between stress and cardiovascular reactivity due to differences in defensive hostility used median splits on the Cook-Medley

and Marlowe-Crowne scales (Shapiro, Goldstein, & Jamner, 1995). Using a slightly different cold pressor task than that of Mente and Helmers (1999), 106 male and 103 female college students (aged 17-33 years) exhibited differential diastolic blood pressure reactivity based on defensiveness, but the hostility X defensiveness interaction overall was not significant. The explanation provided by Mente and Helmers may also be applied to this study, in that the cold pressor task may have failed to replicate the more stressful or anger provoking elements of other challenges.

Summary: anger, hostility, defensiveness associations with cardiovascular reactivity

In summary of studies relating anger, hostility, and defensiveness to cardiovascular reactivity, five studies showing a positive relationship between anger and hostility and cardiovascular reactivity have been reviewed. Such results are not found across the board, as was noted in review of four studies that failed to find an anger/hostility and cardiovascular reactivity relationship. Some researchers have attempted to account for such findings by examining other variables such as defensiveness and inhibition. Four such studies showing a relationship between inhibition/defensiveness, anger/hostility, and cardiovascular reactivity were discussed, followed by descriptions of two studies that failed to find a role of defensiveness/inhibition in interaction with anger or hostility. In both of the negative studies, however, the authors suggested that their methods or participant selection approaches might account for the failure to reveal the relationship.

Anger, Hostility, Defensiveness/Repression and Myocardial Ischemia

The previously reviewed studies noted above linked the personality and

behavioral characteristics of anger, hostility, and defensiveness to cardiovascular reactivity. These studies complement other studies, such as those noted by Rozanski, Blumenthal, and Kaplan (1999) linking anger and hostility to the pathogenesis of cardiovascular disease. Other research has inspected the potential roles of anger, hostility, and defensiveness in acute stress responding among patients with cardiovascular disease using ischemia, rather than CVR, as the dependent measure. Some of these studies have been mentioned before, as researchers have presented findings related to both reactivity and ischemia, however, this section of the proposal will review these studies focusing on the ischemia measures as they relate to psychosocial measures of anger, hostility, and defensiveness.

Anger and hostility associated with myocardial ischemia

Mental stress-induced ischemia has been shown to be predictive of future adverse cardiovascular events among patients with cardiovascular disease (Krantz et al., 1999; Jiang et al., 1996; Jain, Burg, Soufer, & Zaret, 1996). Studies regarding the roles of anger and hostility have shown they are associated with ischemia in acute stress situations. Burg, Jain, Soufer, Kerns, and Zaret (1993) conducted a study of 30 CAD patients (male to female ratio not reported beyond stating sample was predominantly male) and found that among the 15 patients exhibiting ischemia in response to mental stress (mental arithmetic), there were higher levels of hostility, aggressive responding, and trait anger as well as lower anger control as compared to the non-ischemic patients. Ischemia was assessed via radionuclide ventriculography (a decrease of greater than or equal to 5 percent of ejection fraction) and by ECG (ST segment depression greater than or equal to 1 millimeter). The Structured Interview was used to measure behavioral reactivity (a

pattern of behaviors manifest during the interview), which was positively associated with duration of left ventricular dysfunction during the stress task. The Cook-Medley Hostility scale assessed aggressive responding, hostile affect, and cynicism. No group differences were noted for the cynicism factor, but the ischemia with mental stress group scored significantly higher in aggressive responding and hostile affect. The ischemic group was also significantly higher in trait anger, but no differences were noted for Anger-In or Anger-Out. Thus in this study, the authors found that those who exhibit mental-stress provoked ischemia were more emotionally reactive to social interaction and mental provocation, with anger being their primary affective state as assessed by the Structured Interview. Anger expression was not assessed, however, in response to the ischemia-inducing mental arithmetic task.

Another set of studies found that hostility was associated with more severe ischemia among middle-aged men and women (Helmers et al., 1993). In one study, the researchers assessed myocardial perfusion with thallium exercise scintigraphy during a standard Bruce protocol exercise treadmill test among 80 CAD patients. Statistically controlling for gender effects, Composite Hostility was positively correlated with severity of perfusion defects. Analysis by gender revealed that across the 63 male participants this relationship was nonsignificant, but was significant among men aged less than 60 years. The positive correlation between perfusion deficit severity and Composite Hostility was also significant among the smaller set of 17 women. In a second study with an overlapping sample, 42 of the patients underwent 24 to 48 hour ambulatory ECG Holter monitoring. After controlling for gender, both Cook-Medley hostility and Composite Hostility were positively correlated to total minutes of ischemia. A significant gender X

hostility interaction was also manifest, with these relationships stronger among women. This research supports the argument that hostility's pathogenic influence seems to occur at an earlier disease stage, in that among the men, the effect is only manifest in those under the age of 60, and among women, who appear to be protected from CAD development until reaching menopause, and thus may be at an earlier disease stage.

The emotion of anger was reported as a trigger of myocardial ischemia by Gabbay et al. (1996), with ischemia being nearly twice as likely to occur during times of high self-rated anger compared to times of low self-rated anger. Patients with CAD and evidence of out-of-hospital ischemia underwent ambulatory ECG monitoring for 24 to 48 hours while maintaining a validated structured diary regarding physical and mental activities and psychological state. While patients spent the largest proportion of their time engaging in low intensity physical and mental activities, the likelihood of ischemia was greatest during intense physical and stressful mental activities, such that when adjusted for time spent at each level of mental activity, significantly more ischemia was found to have occurred during the highest intensity of mental activity. In dichotomizing anger ratings (from 0, 1 and 2 = "low" vs. 3 and 4 = "high"), a logistic regression showed a trend toward significance for the effect of anger on the likelihood of ischemia (p <0.057). Ischemia was nearly twice as likely to occur when participants' self-ratings of anger were high as when self-ratings of anger were low. This analysis was for "silent" ischemia, and thus excluded cases where ischemia was accompanied by angina, which might have influenced participants' reporting. This study and the previously discussed study (Helmers et al., 1993) present evidence of the role of hostility and anger in daily life, as opposed to in the laboratory such as in the work of Burg and colleagues (Burg et al.,

1993).

Verrier and colleagues presented a canine model of anger to discuss how acute anger may lead to ischemia (Verrier, Hagestad, & Lown, 1987). Their research revealed that post-event ischemia with profound coronary vasoconstriction developed two to three minutes after the elicitation of an anger-like response. The dogs in this study were exposed to a confrontation protocol centered on access to food. After an overnight fast, a leashed dog was first presented with food that was then moved just out of reach. A second leashed dog was then introduced and allowed to consume the food. Under these circumstances, the first dog almost always exhibited an anger-like behavioral response, including growling and exposing teeth. While the dogs were never in physical contact with each other, the anger response continued as long as the animals could still see each other. The behavioral anger response was characterized physiologically by consistent increases in heart rate, mean arterial blood pressure, coronary blood flow, and plasma catecholamine levels. The increased coronary blood flow is necessary to respond to the increased demand on the heart with the anger response. The physiological changes noted in the canine model, such as increases in heart rate, mean arterial pressure, and coronary blood flow, are similar to those noted in humans, and by extension it may be suggested that ischemia is elicited among vulnerable humans (those with CAD) as well.

Legault, Langer, Armstrong, and Freeman (1995) found no significant relationship between Structured Interview assessment of hostility and prevalence of ambulatory ischemia in a sample of 46 patients with stable coronary artery disease. In their study, they inspected the ability of ischemic responses from exercise stress testing and mental stress testing to predict silent myocardial ischemia during ambulatory

monitoring. Half of the patients (23) had an ischemic response to the mental stress of giving a speech concerning personal faults (Rozanski et al., 1988), and this ischemic response to mental stress was a significant predictor of ischemia during a 48 hour period of Holter monitoring independent of response to exercise. Further, the participants with mental stress-induced ischemia had more frequent episodes of ambulatory ischemia with a longer total duration of ischemia during ambulatory monitoring. The failure to find a relationship between ambulatory ischemia and hostility in this study compared to positive findings of hostility related to ischemia such as in the work of Burg and colleagues (1993) and Helmers and colleagues (1993), may be attributed perhaps to the use of the Structured Interview assessment of hostility rather than use of the Cook-Medley. However, it is important to note that this SI hostility assessment was predictive of MI or coronary death in an earlier study (Dembroski, MacDougall, Costa, & Grandits, 1989). Dembroski and colleagues' research in the MRFIT study indicated that the relationship between hostility and CAD was manifest in relatively younger populations, with SI hostility predicting CAD endpoints among those who were 47 years of age or younger over an approximately 7 year follow up, but not among those older than 47 years of age.

Defensiveness and myocardial ischemia

Repression, or defensiveness, in combination with hostility and anger, has also been linked to ischemic events. Helmers et al. (1995) reported a set of studies demonstrating that the combination of high hostility as measured by the Cook-Medley Hostility Inventory and high defensiveness as measured by the Marlowe-Crowne Social Desirability Scale (the two combined being termed Defensive Hostility) was indicative of more functionally severe ischemia in a population of CAD patients. This study showed

the relationship between defensive hostility and ischemia in three different approaches. In one study, defensive hostile participants, compared to Hostile only, Low Hostile, and Defensive only participants, demonstrated the greatest perfusion defects as measured by exercise thallium scintigraphy. In a second study, defensive hostile patients had the most frequent episodes of ischemia during ambulatory electrocardiographic monitoring for 24 to 48 hours. Lastly, in a laboratory study, the defensive hostile participants exhibited the most severe mental stress-induced ischemia as assessed by echocardiography. This is the only study presented to date regarding the relationship between ischemia and defensive hostility, or repressed hostility.

Summary: Anger, hostility, defensiveness, and myocardial ischemia

This component of the proposal has reviewed a few prior studies regarding the associations of anger, hostility, and defensiveness to ischemia. Two studies linking hostility to ischemia were presented (Burg et al., 1993; Helmers et al., 1993), and a study linking acute anger to ischemia was presented (Gabbay et al., 1996). One study noted no relationship between hostility and ischemia, but used a different measure of hostility than did the other ischemia studies (Legault, Langer, Armstrong, & Freeman, 1995). Lastly, the link between hostility and defensiveness related to ischemia was presented in a single study (Helmers et al., 1995).

Overall, while the evidence for an association between hostility and ischemia accumulates, the finding of defensive hostility being associated with ischemia has not been widely replicated. In discussing the studies, a canine model for anger leading to ischemia was briefly presented (Verrier, Hagestad, & Lown, 1987). The next section addresses other models relating anger, hostility, and defensiveness to coronary artery

disease.

Theoretical models, mechanisms relating anger, hostility, and defensiveness to CAD

There have been a number of models presented in explanation of the relationships found between anger, hostility, and defensiveness. Five of the most commonly presented models will be presented at this point.

In a meta-analytic review of research on hostility and physical health, Miller and colleagues (Miller, Smith, Turner, Guijarro, & Hallet, 1996) described three commonly presented models for understanding how anger and hostility might be related to cardiovascular disease endpoints. The psychosocial vulnerability model suggests that adults with hostility may be vulnerable to CAD due to a combination of low levels of social support and high levels of intrapersonal distress. These adults also experience more stressful life events because of the interpersonal difficulties associated with hostility and subsequent anger. This model thus presents at least two ways hostility might impact physical health (directly and indirectly). First, hostility confers risk directly as a combination of low levels of social support and high levels of stress and interpersonal conflict result from the individual's hostile interactions with others. Likewise, poor social support and interpersonal conflict may increase hostility expression in a positive feedback loop, further increasing poor social support and conflict. Secondly, there is the possibility that hostility can lead to depression and anxiety. The resulting depression and anxiety then lead to cardiovascular and neuroendocrine changes that facilitate the development of coronary artery disease (CAD; see Musselman, Evans, & Nemeroff,

1998).

The psychophysiological reactivity model is one alternative model that focuses on the role of heightened sympathetic reactivity in linking hostility and disease (Williams, Barefoot, & Shekelle, 1985, in Miller et al., 1996). Specifically, researchers espousing this model hypothesize that frequent anger episodes produce elevated levels of cardiovascular and neuroendocrine responses. These elevated responses then contribute to CAD. However, Miller and colleagues (Miller et al., 1996) reference a meta-analytic study by Suls and Wan (1993) that suggested that while there is evidence for a relationship between hostility and cardiovascular reactivity, the effect is small and may only be manifest in settings involving social stress such as provocation.

The transactional model is presented as Smith and colleagues' solution to integrating and extending the psychosocial reactivity and physiological reactivity models (Smith, 1994; Miller et al., 1996). From this model, "hostile cognitive-emotional states are expected to lead to antagonistic and aggressive behaviors that produce intrapersonal conflict and hostility from others, which, in turn, leads to a reduction in social support and more negative affect" (Miller et al., 1996, p.342). Thus the hostile attitudes create a hostile environment and thereby increase the stress experienced by the individual in a feedback loop. A function of this hostile environment is increased frequency of stressful interactions and conflicts. Regarding the cardiovascular reactivity component of the transactional model, Smith and colleagues posit that hostile individuals experience increased physiologic reactivity under stress, including self-imposed stress. Hence, hostile individuals experience more stressful interactions compared to non-hostile individuals and they also experience more frequent elevations in cardiovascular

responding compared to non-hostile individuals.

A fourth model, referred to as the constitutional vulnerability model (Krantz & Durel, 1983), suggests a different mechanism to explain the relationship between hostility and health. This model asserts that basic biological individual differences cause and explain the psychological and behavioral manifestations of anger, hostility, and aggression. According to the model, the same underlying biological factor(s), such as a hyperresponsive sympathetic nervous system, also confers vulnerability to cardiovascular disease. Thus, rather than presenting one as resulting from the other, this model suggests that both the anger and hostility characteristics and the biological responding characteristics are a result of a third (underlying) biological factor.

Brosschot and Thayer (1998) presented a fifth model, the anger inhibition/vagal inhibition model, for understanding the link between hostility and cardiovascular disease that differs from the previous models linking anger expression, cardiovascular reactivity, and sympathetic tone. This model proposes that in social situations, people inhibit their anger more frequently than they overtly express their anger, regardless of preferred expression style. Inhibition of overt anger expression may be associated with physiologic responding similar to that of overt expressions of anger, however, Brosschot and Thayer (1998) suggest the cardiovascular responding of the anger-inhibiting person does not recover as quickly as does the responding of a person who expresses his anger and moves on. While the vagal tone and cardiovascular recovery component of their model is beyond the scope of this discussion, the anger inhibition component links directly to our previous considerations of anger suppression, repression, and defensiveness. This perseveration of internal upset may be due to rumination or failure to resolve the anger-

inducing problem, referred to by Brosschot and Thayer as “unfinished action tendencies” (Brosschot & Thayer, 1998). Thus, they argue that while physiologically responding in a similar manner to those who become overtly angry and hostile, those who suppress or inhibit expressions of anger do not enjoy the benefits of a release of anger through its expression. For anger inhibitors then, the physiological effects of the anger experience are maintained for a longer period of time, during which time such individuals who also suffer from cardiovascular disease are at increased risk of adverse events (Brosschot & Thayer, 1998).

In summary, five theoretical models have been presented to explain relationships between anger, hostility, and defensiveness and cardiovascular disease. The psychosocial vulnerability model proposes that hostile adults are more vulnerable to CAD due to a combination of low levels of social support and high levels of interpersonal distress. The hostility directly and indirectly leads to cardiovascular and neuroendocrine changes that facilitate the development and exacerbation of CAD. The psychophysiological reactivity model suggests that heightened sympathetic nervous system reactivity due to hostility leads to elevated levels of cardiovascular and neuroendocrine changes that contribute to CAD. The third model, the transactional model, combines the two previous models, considering the importance of both social environment and support and heightened sympathetic reactivity, suggesting that hostile individuals experience more stressful interactions, and more frequent elevations in cardiovascular responding relative to non-hostile people. The constitutional vulnerability model suggests basic biological individual differences account for both the personality and behavioral characteristics of anger, hostility, and defensiveness, as well as the hyperresponsive sympathetic nervous system,

thus a third underlying variable accounts for both sides of the relationship considered by this current study. Lastly, an anger inhibition/vagal inhibition model has been more recently proposed. This model has yet to be studied in depth, but it suggests an expanded role for the parasympathetic nervous system, with poor vagal tone combining with the inhibition of anger leading to slower recovery from cardiovascular demands, and thus expanding periods of acute cardiac vulnerability. All five of these models consider similar elements of personality, behavior, and physiological responding, with varying degrees of emphases placed on each of those elements by the different models.

Rationale

Hostility has been proposed as a factor in development and exacerbation of cardiovascular disease. Stress responding has been presented as a way in which hostility may affect cardiovascular disease (e.g. Hardy & Smith, 1988, Benight et al., 1997). However, there is still uncertainty regarding the extent to which measures of trait hostility predict cognitive, behavioral and physiological responding in patients with cardiovascular disease. One of the assumptions which has not been thoroughly tested is that patients high in the trait measure of hostility, due to their interpretations of their environment, experience an increased frequency and intensity of anger episodes (Brosschot & Thayer, 1998).

A second assumption is that this hypothesized increase in anger in turn leads to an increase in physiological stress responding, which is often assessed in terms of basic cardiovascular reactivity and ischemia among this vulnerable population (e.g. Anderson & Lawler, 1995, Helmers et al., 1993). Whether or not anger is an essential element for

hostility to lead to increased cardiovascular reactivity and ischemia-- if anger mediates the effect of hostility on cardiovascular reactivity and ischemia-- has not been fully demonstrated.

To account for conflicting findings on the relationships between the psychological and personality variables of anger and hostility and CAD-related variables (to include cardiovascular reactivity and ischemia) some researchers of the relationship between hostility and anger have focused on social desirability or defensiveness (Helmers et al., 1995, Siegman, 1994), postulating that certain highly hostile people are also concerned with preserving self-esteem or presenting themselves in a positive light. Such individuals then must suppress or repress their anger to continue to present themselves positively. Researchers suggest that such individuals might report lower levels of negative emotions such as anger, but that their negative emotions continue to be manifest at a physiological level in the absence of the verbal report of the anger. This would seem to suggest that in highly defensive individuals, anger reports would be poor predictors of cardiovascular reactivity, or in a highly defensive cardiovascular disease population, anger reports would be poor predictors of acute measures of changes in myocardial function such as ischemia. The role of defensiveness in the hostility-cardiovascular disease relationship, while postulated by previous researchers, has not been conclusively determined- it is not clear whether defensiveness mediates the effect of hostility on either cardiovascular reactivity or ischemia.

HYPOTHESES

In the present study, there are three main hypotheses. The first considers the hostility/anger relationship among patients with cardiovascular disease, the second considering the possibility of anger expression mediating the relationship between hostility and the physiological variables of cardiovascular reactivity and ischemia, and the third concerns the possible mediating role of defensiveness/repression in the relationship between hostility and the variables of cardiovascular reactivity and ischemia.

1. To test the assumption that high hostility is associated with an increased intensity of anger expression among patients with cardiovascular disease, it is hypothesized that:

Among these patients, higher Cook-Medley Hostility scores will positively correlate with greater anger self-reports in response to mental challenges of mental arithmetic, anger recall, and the Stroop Color-Word task.

2. To test the assumption that hostility positively correlates with increased cardiovascular reactivity and ischemia due to its influence on anger expression, it is hypothesized that:

Anger expression in response to mental challenge tasks mediates the effects of hostility on cardiovascular reactivity and ischemia. Those with hostility combined with an anger expression response to the mental challenges will respond with greater cardiovascular reactivity (as assessed by blood pressure and heart rate measures) and more frequent ischemia.

Lastly, one may consider the potential role of defensiveness and repression in the hypothesized hostility/anger expression relationship and the hypothesized hostility/physiological reactivity relationship. This consideration leads to the last hypothesis. It is expected that highly defensive participants would express lower levels of anger, yet still have a strong cardiovascular response to a mental stress challenge. Consistent with the conceptualization of defensive individuals as concerned with a positive self-representation and with a need to be viewed in a positive manner by others, it is also expected that defensive participants will report higher levels of interest in the tasks. Thus, hypothesis three concerns relationships of defensiveness to self-ratings, cardiovascular reactivity, and myocardial ischemia.

3. Greater defensiveness (as assessed by the Marlowe-Crowne Social Desirability Scale), will be associated with:

a) lower levels of expressed anger and higher reported interest levels in response to mental challenge and
b) greater cardiovascular reactivity and more frequent ischemia in response to mental challenge (due to the "mental effort" or increased burden of positive self-presentation under challenging circumstances).

Further, it is hypothesized that:

c) defensiveness will mediate the effects of hostility on cardiovascular reactivity and myocardial ischemia. Those with hostility combined with defensiveness will respond with greater

cardiovascular reactivity and greater myocardial ischemia.

In summary of the three main hypotheses, the first hypothesis targets the hostility-anger relationship during mental challenges among cardiovascular disease patients. The second hypothesis addresses the importance of anger expression in the proposed hostility/cardiovascular responding relationship. The last hypotheses explore the influence of defensiveness on verbal reports and physiological responding and the potential role of defensiveness as a mediator in the hostility/cardiovascular responding relationship.

METHODS

Design and Overview

Participants

Study participants took part in a larger study examining the role of physical and mental activities on cardiovascular symptoms, ischemia, and blood flow (the Triggers of Myocardial Ischemia Study: TOMIS). Male and female patients with suspected cardiovascular disease were enrolled if they had a recent prior positive exercise test or typical anginal complaints, and were referred for elective diagnostic cardiac catheterization at Georgetown University Hospital, Walter Reed Army Medical Center, or the Washington DC Veterans Affairs Hospital. Participants gave their informed consent, and were informed that they would be asked to complete some mental and physical tasks that simulate daily activities while their hearts were to be monitored closely by various instruments.

Inclusion criteria were age less than 80 years, signs of inducible myocardial ischemia or anginal symptoms suggestive of CAD, left ventricular ejection fraction (LVEF) greater than 30 percent, capable of informed consent, and no baseline abnormalities of their electrocardiogram interfering with ST-segment analysis including use of digitalis. Exclusion criteria included refusal to consent, recent acute myocardial infarction (one month), psychiatric disorder interfering with mental stress or informed consent, recent angioplasty (6 months), significant congestive heart failure (CHF), unstable angina, critical valvular pathology, severe disease of the left main coronary artery, and cardiomyopathy.

Patients participating in the laboratory phase (with the radionuclide ventriculography component, labeled RNV) and completing the psychological questionnaires were eligible for the current study (please refer to B. Procedures, below). Seventy-two patients completed the laboratory phase of the protocol, with 59 of those participants also validly completing the psychological questionnaires. Among those 59 participants, data were available in varying amounts for anger and interest ratings, cardiovascular reactivity, and myocardial ischemia. Fifty-six individuals provided anger and interest ratings. Fifty-eight participants had cardiovascular reactivity data recorded for at least one of the mental stress tasks. Forty-seven participants had interpretable radionuclide ventriculography imaging data available for ischemia assessment. The rationale for the current study to examine coronary artery disease patients rather than healthy individuals is that it allows us to investigate both cardiovascular reactivity and myocardial ischemia. Myocardial ischemia with mental stress is rarely manifest among healthy persons (less than 5 percent) and occurs in 30-60 percent of patients with coronary artery disease (Rozanski & Berman, 1987; Krantz et al., 1996).

Procedure

The current study examined a subset of the measures and sessions of the TOMIS protocol. TOMIS participants participated in 1) a laboratory phase with mental stress tasks (and RNV), 2) questionnaires, 3) a cardiac catheterization phase with a mental stress task, and 4) a phase of ambulatory ECG monitoring concurrent with completion of an activity diary. The current study integrated the first two components, the laboratory phase and the questionnaires relevant to assessment of hostility and defensiveness.

The laboratory protocol involved a rest period followed by the three mental stress

tests being presented in counterbalanced fashion with rest periods between tasks allowing the participants' physiological responding to return to resting or baseline levels. Concurrent with the rest periods and mental stress tasks, measures of cardiovascular reactivity via Dynamap, and of ischemia via radionuclide ventriculography were being taken. Following the tasks a debriefing was performed to more fully explain the nature of the mental stress tasks to the participants, and answer any concerns they might have had.

In the TOMIS protocol, the order of participation in the cardiac catheterization phase and the laboratory protocol phase varied among participants, thus participants participating initially in the cardiac catheterization phase were no longer naive to the mental arithmetic task (described below) when they completed the laboratory protocol. Of the 69 participants completing both phases, 22 participants completed the laboratory phase prior to the cardiac catheterization (47 participants completed catheterization prior to the laboratory protocol).

The Cook-Medley Hostility Scale and the Marlowe-Crowne Scale were administered in conjunction with other questionnaires prior to the laboratory testing or within the week of the procedure. The participant was provided copies of the questionnaires with a brief explanation and instructions as to how to complete the measures. The participant completed the questionnaires outside of the laboratory setting and returned the questionnaires either in person or by mail.

Mental Stress Testing

Mental Arithmetic Task

Participants were instructed that their performance would be rated for speed and accuracy, and to try as hard as they could. Participants were instructed to listen to a standardized audiotape that begins with instructions for the task, which are for the participant to verbally subtract serial 7's from a set of specified four digit numbers for a period of five minutes (Krantz et al., 1991). After the instructions on the audiotape were played, the tape was stopped and the experimenter asked if the participant had any questions, following which the tape restarted. The voice on the tape indicated the specific number from which to count, and a loud metronome sound was also reproduced on the tape. As the participant progressed through the task, the experimenter interrupted and harassed the individual with prompts such as "You're not trying hard enough. Concentrate!" and "accuracy and speed are important" (Krantz et al., 1991).

Anger Recall Task

As part of the initial orientation to the study, the experimenter solicited from the participant a recent episode during which the participant became extremely irritated, frustrated, angry or upset. The participant then was thanked and told that he/she may be asked to comment on that event later. At the start of the actual task, the experimenter reminded the participant of the anger-provoking episode, and following some comments directing the participant to mentally place themselves back in the situation, the participant was asked to make a five minute speech about the incident, to recreate the situation, talking about the events, especially regarding the participant's thoughts and feelings during the event. The experimenter audiotaped the speech, holding or placing the audiotape recorder in close proximity to the participant. The experimenter at times

during the speech would briefly prompt and redirect the participant if he failed to continue his speech or became distracted and his speech became less relevant to the anger recall task (Ironson et al., 1992).

Stroop Color-Word Task

The computerized Stroop Color-Word task was presented in an automated manner via personal computer. The computer was placed in view of the subject, and he/she was provided the computer mouse/keyboard to indicate responses to the task. The task was programmed to present stimuli and adjust its level of difficulty and pacing of the Stroop task to the participant's level of performance (Legault et al., 1991; Stroop, 1935). Subjects were presented with a list of color words on the monitor (blue, green, red, etc.) each word being presented in a color different than the color designated by the word (for example, the word "blue" is presented in red, green, or yellow). The subject's task was to designate the color in which each word is presented as quickly as possible. This program was implemented in the Psychophysiological Investigations of Myocardial Ischemia Study (PIMI; Becker et al., 1996; Goldberg et al., 1996).

Assessment of Psychological Variables

The hostility and defensiveness questionnaires were provided for the participants to complete outside of the laboratory setting during the week prior to or the week following the laboratory session. Acute anger and interest ratings were taken following a baseline resting period just prior to tasks, and then again immediately following each task.

Hostility

The Cook-Medley Hostility Inventory is a true/false questionnaire of fifty items.

Scores may range from 0 to 50, with items endorsed as "true" counting as one point (with 3 items reverse scored). The Cook-Medley Hostility (Ho) Scale was derived from the Minnesota Multiphasic Personality Inventory (Cook & Medley, 1954). This hostility scale was initially developed to differentiate between teachers having good versus bad rapport in their relationships with students, but has become popular among health outcomes researchers as the evidence accumulates suggesting it is an important predictor of future cardiovascular health status (see Smith, 1992 for review). The Cook-Medley Hostility Scale contains 5 subscales reflecting negative attitudes towards life in general. Those five subscales are Cynicism, Hostile Affect, Aggressive Responding, Hostile Attitude, and Social Avoidance, and a Composite Hostility Score may be obtained by summation of the first three subscales. Descriptive findings in the literature vary. Barefoot et al. (1989) found a mean Ho score of 20.8 (± 0.77) for a 1985-1986 sample of undergraduate students, and a mean of 22.3 (± 0.85) in a 1986-1987 undergraduate student sample. Helmers and Krantz (1996) reported a mean of 21.03 (± 6.9) in their study of 68 healthy men and women. Helmers et al., (1993) reported a mean of 16.9 (± 5.9) in a sample of 63 men with CAD and a mean of 14.6 (± 8.6) in a sample of 17 women with CAD. The questionnaire as provided to the participants is included in the appendix.

Defensiveness

Defensiveness is operationalized as participants' total scores on the Marlowe-Crowne Social Desirability Scale. The Marlowe-Crowne Social Desirability Scale is a true/false questionnaire of 33 items that measures defensiveness. Scores may range from 0 to 33, with items endorsed as "true" counting as one point towards the total score (with

fifteen items reverse scored). Items in the scale reflect socially desirable behaviors or reflect cognitions that are untrue of almost all people (Crowne & Marlowe, 1964). Defensiveness may be defined as the unconscious or conscious tendency to not report socially undesirable aspects of oneself, having a concern to represent oneself in a positive manner, and in so doing be seen in a positive manner by others (Crowne & Marlowe, 1964; Paulhus, 1984). Recent studies have reported varying descriptive data. Al'Absi, Bongard and Lovallo (2000) reported a median score of 16 on the Marlowe-Crowne Social Desirability Scale. In comparing normotensive and mild and severe hypertensives, Mann and James reported means of 15.4 (± 5.1), 17.5 (± 5.9), and 19.2 (± 4.5) respectively (1998). A sample of 61 male CAD patients had a mean Marlowe-Crowne score of 18.0 (± 5.5), and a sample of 17 female CAD patients had a mean of 19.0 (± 4.1; Helmers et al., 1995). The Marlowe-Crowne Social Desirability Scale was combined with another measure in the questionnaires provided to participants. An example of that form is provided in the appendix.

Anger Expression & Interest

Just following a baseline resting period, participants were asked to rate how they are feeling just prior to the rating on the topics. For the Likert-type scales, the participants are asked to rate on a scale of 1 (not at all) to 7 (very much), the amount that each item in a list of feelings applies to them. Among the items are "interested" and "angry." Other affect ratings in the larger study include those of "chest pain, anxious, irritated" and "tired." They are asked to make these ratings following each mental stress task in addition to the baseline rating. This type of rating has been implemented as part of a validated diary system (Krantz et al., 1996; Kop et al., 2001). Samples of the forms

recording responses are included in the appendix.

Responses to Mental Stress

Blood pressure and heart rate

During the laboratory protocol of rest and mental stress tasks, an automated Criticon Dynamap blood pressure cuff was attached to the participants' nondominant arm (e.g. Helmers & Krantz, 1996). The participant was instructed to refrain from moving their arm while the cuff inflated and monitored blood pressure. Systolic and diastolic blood pressure and heart rate measurements (SBP, DBP, and HR) were taken every two minutes throughout the protocol. Resting blood pressure and heart rate levels were quantified as the means of the last three consecutive readings taken during the rest period. Aggregate baseline measures of SBP, DBP, and HR for each participant were developed by calculating the mean of resting baseline measures across the three tasks. During the stressors, blood pressure and heart rate measures were quantified as mean scores for each of the stressors that were residualized for baseline based on previously described procedures (Manuck, Kasprowicz, Monroe, Larkin, & Kaplan, 1989). To reduce measurement error, Kamarck et al's procedures to increase test-retest reliability were implemented (1992). Blood pressure (BP) and heart rate reactivity were determined in two ways in the subsequent analyses, one method being calculation of simple change scores between the aggregate baseline and the mean of the measures during the specific tasks. The second method, employed in some of the regression analyses, involved employing the aggregate baseline measure as a covariate, with the mean of the measure during the specific task serving as the dependent variable.

Myocardial Ischemia

Ischemia was assessed during the laboratory protocol via radionuclide ventriculography. R-wave synchronized, multiple-gated equilibrium radionuclide ventriculography was performed with a gamma camera positioned in the left anterior oblique angle (to optimally separate left and right ventricles). During rest periods and the mental stress tasks, multiple-gated blood-pool images (MUGA images) were taken in three-minute acquisitions, with the images collected at 16 frames per second. One MUGA image was taken at the end of each rest from minutes 12 to 15. One image is selected per stress task.

The images of the left ventricle provide information regarding wall motion abnormalities indicative of ischemia, and also provide information regarding ejection fraction. Wall motion was assessed in patients by consensus of two experienced observers blind to clinical data and image order. Wall motion was scored on a five-point scale, with a score assigned to each of five ventricular segments (basal, septal, apical, inferior, and posterolateral) using the following classification system: 3 = normal motion, 2 = mild hypokinesia, 1 = moderate to severe hypokinesia, 0 = akinesia, and -1 = dyskinesia. Determination of wall motion worsening was made when the score decreased by at least 1 from both baseline and the immediately preceding score. A wall motion summative score can then be generated by adding the parameters of extent (number of segments worsening) and severity (magnitude of the individual segments worsening; Krantz et al., 1991).

Three laboratories provided radionuclide ventriculography analysis. Twenty participants' results were analyzed at Cedars-Sinai Medical Center, 22 participants'

results were analyzed at Georgetown University Medical Center, and 17 participants' results were analyzed at St. Luke's-Roosevelt Medical Center (twelve laboratory location unreported/report missing). Where multiple laboratories provided analysis (12 cases), the report was selected by completeness and quality of report. Nine cases were analyzed by both Cedars-Sinai and St. Luke's-Roosevelt. Three cases were analyzed by both Cedars-Sinai and Georgetown University laboratories. In final analysis, 20 reports were utilized from Cedars-Sinai Medical Center, 19 were provided by Georgetown University Medical Center, and eight were provided by the laboratory from St. Luke's-Roosevelt Medical Center.

Analyses were conducted to examine the possibility of differences between participants whose data were analyzed at Cedars-Sinai as opposed to Georgetown or St. Luke's-Roosevelt regarding independent measures (related to hostility, anger, and defensiveness) as well as participant demographics, risk factors, and clinical characteristics. No differences between groups were detected for the hostility, anger, and defensiveness measures. The only notable difference between the groups for other factors was with education level, with participants whose data were analyzed at Cedars-Sinai being less educated compared to others [10.6 ± 3.4 years of education versus 13.6 ± 3.0 years; $t(48)= -3.276$, $p = .002$].

Statistical Analyses and Power Analyses

Statistical Analyses

Hypothesis 1. To determine the role of hostility in predicting anger expression, first, product moment correlations and multiple regression analyses were conducted with

Cook-Medley Composite Hostility and aggregate baseline anger ratings serving as the predictor variables and the post- task Likert-type ratings of anger (anger expression) as the criterion variable. An analysis was conducted for each of the three tasks (Math, Anger Recall, and Stroop) individually.

Another analysis was conducted to predict peak anger rating across the three tasks, with Composite Hostility as the predictor, adding in aggregate anger expression baseline as a predictor, and using aggregate peak anger expression as the criterion variable. Aggregate anger expression baseline was calculated as the average of anger ratings across the three baselines. Aggregate peak anger expression was the value of the highest anger rating post-task among the three tasks.

A third set of analyses examined components of hostility, specifically, Barefoot et al.'s (1989) Composite Hostility components, cynicism, hostile affect, and aggressive responding, and aggregate baseline anger expression as predictors of post-task anger expression. Each of the three components served with aggregate baseline anger ratings as predictors for post-task anger ratings. A last step in each of the three sets of analyses was an adjustment for the potential confounders of age and gender, and any other participant demographic or clinical characteristic that was significantly associated with hostility and anger ratings.

Two independent samples t-tests were conducted to assess the possibility of order effects of the laboratory protocol on questionnaire responding. One t-test compared means of the Cook Medley Hostility scale for participants completing the questionnaire prior to the laboratory protocol to the means of participants completing the questionnaire following the protocol. A second t-test inspected means for the Marlowe-Crowne Scale

of Social Desirability in a similar manner (participants completing the questionnaire before versus after the laboratory protocol).

Hypothesis 2. The second set of hypotheses was examined by assessing whether the effects of hostility on cardiovascular reactivity and ischemia were mediated by expression of acute anger. To quantify cardiovascular reactivity, two methods were employed: one approach using simple change scores, the second used aggregate baseline cardiovascular measures as covariates within regressions predicting actual levels of the cardiovascular measure during each task.

In these and following analyses, the approach was to examine additive and suppressing effects of mediators. Set-wise hierarchical regression analyses were used to assess the effects of hostility and anger expression on cardiovascular reactivity. Four sets were examined, set one consisting of individual control variables and set two consisting of hostility. Set three consisted of anger expression and the fourth set considered a hostility X anger expression interaction. The first set consisted of the control variables of age, gender, hypertension, disease severity, and the appropriate aggregate baseline cardiovascular measure, as well as any potential confounds identified among participant demographic and clinical characteristics. For the second set, Composite Hostility was entered as the predictor variable for cardiovascular reactivity. The third set considered the effect of anger expression on cardiovascular reactivity by use of aggregate baseline anger and post task anger scores as the predictor variables. The fourth set employed a Composite Hostility X anger reactivity interaction. The anger reactivity measure factored into the interaction term was a residualized anger change score obtained by a regression with pre-task anger as a predictor variable, and post-task anger as the criterion, or

dependent measure. The purpose of these analyses was to determine whether hostility alone is a significant predictor of cardiovascular reactivity, and whether addition of anger suppresses the hostility-cardiovascular reactivity relationship. In combination with the first analysis, these analyses indicated the relative importance of hostility and anger expression.

To consider the relative importance of hostility and anger expression in ischemia, two sets of Chi-square analyses tested for independence between 1) Composite Hostility and stress-induced myocardial ischemia presence/absence and 2) between anger expression changes and the presence of stress-induced ischemia. The bivariate analyses provided by Chi-square do not account for possible covariates. It had been proposed that were Chi-square analyses to reveal a relationship between either Composite Hostility or anger expression changes and the presence/absence of stress-induced myocardial ischemia, then multivariate analyses by setwise multiple logistic regression would then be conducted. Those regressions would employ similar sets of predictor variables as were used in the analysis of cardiovascular reactivity (control variables, Composite Hostility, and anger difference scores) with the dichotomous criterion variable of presence or absence of stress-induced myocardial ischemia. These analyses were intended to parallel those described above and similarly indicate the relative roles of hostility, anger expression, and anger expression after controlling for hostility in ischemia.

Hypothesis 3. To test hypothesis 3a that anger expression and defensiveness were negatively correlated and that interest and defensiveness were positively correlated, a Pearson product-moment correlation was used. The relationship of defensiveness to cardiovascular reactivity and ischemia (hypothesis 3b) was also assessed using Pearson

product-moment correlations.

To assess the role of defensiveness in the hostility/cardiovascular reactivity relationship (hypothesis 3c), setwise hierarchical multiple regression analyses were employed (systolic blood pressure changes, diastolic blood pressure changes, and heart rate changes). Five sets were examined. The first set consisted of individual control variables (age, gender, hypertension, disease severity, aggregate baseline cardiovascular measure, and any potential confounders identified from participant demographic/clinical characteristics). The second consisted of Composite Hostility as the predictor variable for cardiovascular reactivity. The third set consisted of defensiveness as the predictor, and the fourth considered the Composite Hostility X defensiveness interaction. The fifth set evaluated levels of anger expression in predicting cardiovascular reactivity. Analysis of the last set (anger expression) indicates, in conjunction with the anger expression/defensiveness correlation scores, the magnitude of potential overlap between the two measures of anger expression and defensiveness in mediating the hostility/cardiovascular reactivity relationship.

To consider the relative importance of defensiveness in relation to myocardial ischemia (hypothesis 3c), a Chi-square analysis tested for independence between defensiveness and ischemia. The bivariate analyses provided by Chi-square do not account for possible covariates. It had been proposed that were Chi-square analyses to reveal a relationship between Composite Hostility and Defensiveness, then multivariate analyses by setwise multiple logistic regression would then be conducted resembling the sets employed for the examination of the role of defensiveness on cardiovascular reactivity noted above. The dichotomous criterion variable is presence/absence of

ischemia.

Power Analyses

To determine the sample size needed for this study, power analyses for each of the three hypotheses were conducted.

Hypothesis 1. The first hypothesis concerned the relationship between hostility and anger expression. Existing studies involving Cook-Medley Hostility and measures of anger expression following mental challenges were examined. One study found a significant effect of Buss-Perry Aggression Questionnaire Hostility on anger ratings on the Profile of Mood States following a task including experimenter mistreatment, with the high hostility group significantly more angry following provocation [$F(1,31)= 4.4$, $p < 0.05$; Felsten & Hill, 1999]. Participants in another study with high Cook-Medley Hostility scores were significantly angrier on a 7-point Likert-type scale than low hostility participants in a video game competition with harassment [$F(1,63)= 5.5$, $p < 0.05$; Felsten, 1995]. These studies suggested a moderate to large effect size for the relationship between Cook-Medley Hostility and anger measures. Based on these prior studies, indicating correlations of pre-task anger with post-task anger ratings (correlations of approximately 0.4), the estimated squared multiple correlation (R^2) was deemed to be approximately .20 prior to entering hostility. The alpha level for Hypothesis 1 was adjusted for multiple comparisons (3 tasks and one combined "peak" measure) to 0.05/4 = 0.0125. For a multiple linear regression model which already includes baseline anger as a covariate with a squared multiple correlation of 0.20, a sample size of 53 would have 80 percent power to detect significance at a Bonferroni adjusted alpha = 0.0125 in order

to detect an increase in R^2 of 0.15 attributable to hostility as an additional covariate. Over 80 study participants completed the Cook-Medley Hostility questionnaire, with 59 of them also completing the laboratory portion during which they provide anger ratings, providing a sample size for greater than 80 percent power.

Hypothesis 2. Studies relating hostility and anger to cardiovascular reactivity and ischemia suggested a moderate effect size, having shown correlations from 0.28 to 0.55. Cardiovascular reactivity was linked to the Buss-Perry Aggression Questionnaire (AQ; Buss & Perry, 1992) by Smith and Gallo (1999). An interaction of the AQ and an evaluative threat condition accounted for 15.4 % of the variance in SBP reactivity. In linking ischemia to hostility, Helmers et al. (1993) found a 0.42 correlation between Composite Hostility and severity of perfusion defects among 17 women, and among 17 men under the age of 60, a correlation between anger and severity of perfusion defects of 0.55. Further, they reported on an overlapping sample of 42 CAD patients, linking Holter-monitored ischemia and Hostility and Composite Hostility with correlations of 0.28 and 0.29, respectively. Cardiovascular reactivity in this proposed study was assessed by SBP, DBP, and HR responses. The alpha level for this portion of Hypothesis 2 was thus adjusted for multiple comparisons (3 dependent variables) to 0.05/3 = 0.017.

For a multiple linear regression model which already includes 2 sets of covariates (baseline demographics and hostility), with a squared multiple correlation (R^2) of 0.25, a sample size of 56 has 80 percent power to detect, at alpha = 0.017, an increase in R^2 of 0.1250 due to including anger expression as an additional predictor of CV reactivity, matching the obtained sample size.

For the Chi-square test of independence between anger expression and myocardial ischemia, a sample of 55 participants would have 80 percent power to detect the alternative hypothesis of dependence assuming an effect size of 0.2. The obtained sample of 56 participants would thus have greater than 80 percent power to detect a relationship, but uninterpretable or missing data further reduced the obtained sample size to 44, resulting in a power of 69 percent. Were data available for the entire sample, the study would have been sufficiently powered to detect a bivariate association by Chi-square analysis not considering covariates.

Logistic regression analysis is needed to consider covariates, to control for baseline variables and test independence of multiple predictor variables. Logistic regression analyses would examine inducibility of ischemia at a rate of 40 percent. If 74 patients are included in a one-sided logistic regression analysis, after consideration of control variables and Composite Hostility, the power to detect an added risk of 2.5 for ischemia associated with anger expression is 80 percent at alpha = 0.05. A sample size of 56 would have 68 percent power in the same analysis. The odds ratio was based on the assumption of 30 percent ischemia in patients with average anger expression and 52 percent in those with high (mean plus one standard deviation) anger expression. The power based on the available 44 participants is 57 percent.

Hypothesis 3. Studies relating hostility and defensiveness to cardiovascular reactivity and ischemia were reviewed to estimate appropriate effect sizes. Grossman, Watkins, Ristuccia, and Wilhelm (1997) found a 0.45 correlation between Marlowe-Crowne assessed defensiveness and systolic blood pressure reactivity in a sample of patients with stable coronary artery disease consisting primarily of men (42 men, 3

women). In a study relating defensiveness to ischemia, Helmers and colleagues (1995) reported that a first step of a regression model for ischemia predicted 13.3 percent of the variance entering gender, composite hostility, and defensiveness, or a correlation of 0.36. In a second step, an additional 7.3 percent of the variance was accounted for in predicting ischemia, entering a Composite Hostility X Marlowe-Crowne Defensiveness interaction, a correlation of 0.27. Other studies noting significant effects of defensiveness and hostility on cardiovascular reactivity did not report effect sizes (Jamner et al., 1991; Helmers & Krantz, 1996). Such studies suggest a moderate effect size for the relationship between defensiveness and cardiovascular reactivity and ischemia. Consistent with Hypothesis 2, the alpha level for cardiovascular reactivity was adjusted for multiple comparisons (3 dependent variables) to 0.05/3 = 0.017.

For a multiple linear regression model which already includes 3 sets of predictor variables (demographics, hostility, and anger expression) with a squared multiple correlation (R^2) of 0.30, a sample size of 52 has 80 percent power to detect, at alpha = 0.017, an increase in R^2 of 0.1250 due to including defensiveness as an additional covariate to predict cardiovascular reactivity. The obtained sample size of 59 would thus have greater than 80 percent power under those parameters.

For the Chi-square test of independence between defensiveness and myocardial ischemia, a sample of 55 participants has 80 percent power to detect the alternative hypothesis of dependence assuming an effect size of 0.2. Were the study sufficiently powered by Chi-square analysis to detect a bivariate association not considering covariates, logistic regression analysis would still be needed to control for baseline variables and test independence of multiple predictor variables.

Logistic regression analyses would examine inducibility of ischemia at a rate of 40 percent. Given 74 subjects, a one-sided logistic regression analysis, after consideration of control variables and Composite Hostility, would have 80 percent power to detect an added risk of 2.5 for ischemia associated with defensiveness at alpha = 0.05. The expected sample size of 56 has 68 percent power in the same analysis. The power based on the available 44 participants is 57 percent. The odds ratio (as was the same ratio in hypothesis two) is based on the assumption of 30 percent ischemia in patients with average anger expression and 52 percent in those with high (mean plus one standard deviation) anger expression.

Summary

In sum, the number of participants completing the necessary elements of the study yields over 80 percent power for Hypothesis 1 and the cardiovascular reactivity components of hypotheses 2 and 3. The actual obtained sample size for the analyses of the myocardial ischemia components of hypotheses 2 and 3 after adjustment for missing or otherwise inadequate data is less than 80 percent for the chi-square analyses and less than 68 percent for the logistic regression analyses, suggesting logistic analyses will be underpowered, a limitation to be considered in the Discussion section.

RESULTS

Participant characteristics

Questionnaires and laboratory assessments were obtained in 59 patients. Patient characteristics are presented in Table 1. The sample was predominantly male (88 percent), Caucasian (72 percent) with an average age of 61.1 (*SD* =9.0) years. Forty-five patients were enrolled from the Washington D.C. Veterans Affairs Medical Center, nine from Walter Reed Army Medical Center, and five from the Georgetown University Medical Center.

The majority of patients were living with a spouse (68 percent), and had at least a high school education (mean education level 12.5 ± 3.3 years). The majority of participants were not working full time (76 percent), with a large proportion being retired (45 percent).

Cardiovascular risk factors were as follows (Table 2): the sample overall was overweight (Body Mass Index = 28.1 ± 5.2), and the mean total cholesterol level was 204.38 mg/dl (±39.76). Two thirds of the sample was treated for hypertension, and a quarter of the sample reported they currently smoked (see Table 2.). Eleven patients were Type I diabetics (IDDM; 19 percent), and 9 patients were Type II diabetics (NIDDM; 15 percent). Over half of the sample reported a family history of coronary artery disease and almost half of the sample reported a family history of hypertension.

Table 3 displays patients' cardiac history and coronary angiographic information at the time of study. Thirteen participants had previously undergone angioplasty, and two had undergone coronary artery bypass grafting prior to entry in the study. Eighteen

participants had previously suffered myocardial infarctions (31 percent). Forty-nine patients (83 percent) had recorded positive exercise tests prior to participation in the study. The mean ejection fraction for study participants was 61.3 ± 12.3. Patients varied in their New York Heart Association (NYHA) classifications related to angina, with the majority reporting complaints of levels II (18 patients, 31 percent) or III/IV (26 patients, 45 percent). Their average ejection fraction was 61.3 ± 12.3. The current number of diseased vessels for each individual varied, with 15 (25.4 percent) found at catheterization to have 1 diseased artery, 17 individuals (28.8 percent) had 2 diseased arteries, and 18 participants (30.5 percent) with 3 diseased arteries (4 unreported- 6.8 percent).

Effects of cardiac medications were considered, focusing on potential relationships of study variables with beta-blockers, calcium channel blockers, and nitrates. Beta-blockers were prescribed for 23 of 58 participants (one participant's medication status unreported). Fifteen of those participants withheld their medication prior to the mental stress tasks for greater than 36 hours. Beta-blockers were not prescribed for 35 participants. Calcium channel blockers were prescribed for 36 participants, with 31 participants withholding their medication for greater than 24 hours prior to testing. Calcium channel blockers were not prescribed for 22 participants. Nitrates were prescribed for 20 participants, of whom 16 withheld their medications greater than 6 hours. Nitrates were not prescribed for 38 individuals. A one-way analysis of variance was conducted with Composite Hostility, Defensiveness and anger expression change scores for each of the three tasks as the dependent variables with the independent variable of beta-blocker status (3 levels: Not Prescribed, Prescribed but

withheld > 36 hours, Prescribed and not withheld). There was a significant difference in Composite Hostility scores across the 3 groups with post-hoc analyses indicating the group taking beta-blockers, but for whom the medication was not withheld, had lower Composite Hostility scores compared to the other two groups. Closer inspection revealed this effect to be attributed to two outlier low scores in the group. Similar analyses for groups regarding calcium channel blockers (withheld for > 24 hours) and nitrates (withheld > 6 hours) revealed no significant group differences related to Composite Hostility, Defensiveness, or anger expression changes.

Inspection of cardiac medications in relation to cardiovascular reactivity was conducted with repeated measures analyses of variance with the repeated measures of aggregate baseline and during task cardiovascular measures, and the independent variable of medication status (3 levels: Not prescribed, Prescribed but withheld, Prescribed and not withheld). Separate analyses were conducted for beta-blockers, calcium channel blockers, and nitrates. Beta-blocker medication status did not significantly interact with blood pressure reactivity. The interaction between calcium channel blockers and heart rate changes during the Stroop task was significant [$F (2, 41)= 3.85$, $p = .029$], with the participants not prescribed the medication showing less reactivity than the groups for whom calcium channel blockers were prescribed. Nitrates status interacted significantly with heart rate during the Math task [$F (2, 53)= 4.23$, $p = .020$], and during the Anger Recall task [$F (2, 54)= 4.83$, $p = .012$], however the absolute differences in heart rate reactivity across groups were fairly small.

Presence of ischemia during the Math task was marginally associated with nitrate medication ($\chi^2 = 4.787$, $p = .091$), with cross-tabulation suggesting that withholding of

nitrates was associated with more frequent occurrence of ischemia. Nitrate medication status was significantly associated with ischemic status during the Stroop task (χ^2 = 13.405, p = .001), with cross-tabulation suggesting that withholding of nitrates was associated with more frequent occurrence of ischemia. Beta-blocking and calcium channel blocking medications were not associated with presence/absence of ischemia in any of the three tasks in chi-square analyses.

Questionnaire Measures

A total of 59 participants fully completed the Cook Medley Hostility Inventory (see Table 4.). The full-scale hostility mean score for the group was 19.8 (*SD* =9.9). The Composite Hostility mean score (sum of cynicism, aggressive responding, and hostile affect subscales) was 11.9 (*SD* =5.7). A total of 56 participants fully completed the Marlowe-Crowne Scale of Social Desirability to yield a measure of defensiveness (see Table 4). The defensiveness mean was 19.6 (*SD* =6.6). Independent samples t-tests to compare participants' scores for those completing the questionnaire before or following the laboratory protocol suggested no difference between the two groups based on time of administration of questionnaires [Hostility: $t(30) = 0.59$, $p = .56$, mean for those taking questionnaires prior to laboratory protocol = 17.03 ± 9.13, those participating in laboratory protocol prior to questionnaires = 19.27 ± 10.25; Defensiveness: $t(28) = 0.74$, $p = .46$, mean for those taking questionnaire first, 21.80 ± 6.42, the laboratory first, 20.00 ± 6.16]. These t-tests were conducted on the set of participants for whom specific dates of questionnaire completion were available (30 participants).

Anger expression and interest ratings

Anger expression ratings taken before and following tasks suggested that the manipulations were effective in increasing anger expression (see Table 5). Paired samples t-tests indicated a significant effect for all three tasks [Math: $t(57)= 6.50$, $p < .01$; Anger Recall: $t(58)= 7.93$, $p < .01$; Stroop: $t(48)= 4.39$, $p < .01$], with the largest increase in score associated with Anger Recall. Peak anger ratings (for each participant, the highest post-task anger score from among the ratings related to the three tasks) were almost a full point higher than ratings for any one task individually. Interest ratings were much higher during baseline relative to anger ratings, with minimal, or no increases in response to any of the three tasks (see Table 5).

Cardiovascular reactivity

Systolic blood pressure measures taken during the three mental stress tasks were significantly higher than during pre-task baseline periods (increases of 24.1 ± 13.6 mmHg, 20.8 ± 14.0 mmHg, and 21.4 ± 14.0 mmHg for Math task, Anger Recall task and Stroop Color-Word task, respectively; see Table 6., Figures 1-3.). Likewise, diastolic blood pressure, while showing smaller absolute increases, were significantly higher during the task conditions relative to their baselines (increases of 13.2 ± 8.2 mmHg, 12.6 ± 7.0 mmHg, and 10.7 ± 7.4 mmHg for Math, Anger Recall and Stroop respectively). Lastly, heart rate increased significantly during the task conditions, with the largest increase found during the Math task condition (11.6 ± 9.4 beats/minute, or bpm), and the smallest heart rate increase associated with the Anger Recall task (6.1 ± 4.8 bpm). The

number of participants across tasks varies due to technical and practical difficulties, such as computer operation difficulties with the Stroop Color-Word task, or participant's color blindness.

Myocardial ischemia during tasks

Wall motion abnormalities indicative of ischemia (worsening wall motion function greater than one on the five point scale across one or more of the five regions of the cardiac tissue) occurred in approximately 40 percent of the sample across tasks (19 of 47 participants positive; see Table 7.). Ischemia was most frequently manifest during the Anger Recall task (14 of 44, 31.8 percent). The Stroop Color-Word task elicited ischemia in a slightly smaller percentage (9 of 36, 25.0 percent), with participants during the Math task exhibiting the least (9 of 44, 20.4 percent).

Hypothesis 1.

Hypothesis 1 addressed the relationship between hostility measures and anger reports in response to the three stressors of mental arithmetic, anger recall, and the Stroop Color-Word task. It was hypothesized that higher Cook-Medley Hostility scores would positively correlate with greater anger self-reports in response to the three mental stressors.

Initial correlations (see Table 8.) were conducted to examine the bivariate relationships between these anger self-reports and hostility measures. Correlations were calculated with the absolute values (i.e. post-Math anger, post-Anger Recall anger, and post-Stroop anger) and with anger change scores (calculated as the value of post-task

Anger ratings minus the pre-task Anger ratings, labeled "Anger Change Math," "Anger Change Anger Recall", and "Anger Change Stroop"; see Table 8.). Correlations between anger responses by task revealed positive correlations between anger ratings across tasks (see Table 8.), with the Likert ratings of anger (absolute levels) following the Math stressor being correlated with similar ratings following the Anger Recall stressor ($r = .464$, $p = .001$) and the Stroop Color-Word stressor ($r = .358$, $p = .017$). Correlations showed similar but less strong patterns when considering anger change scores associated with each task. Given the varying correlations as well as the anger scores for each of the tasks (Table 5.), it does not appear that these correlations are due to a response set or general response style. The Math and Anger Recall tasks resulted in larger pre- to post-task differences (Math anger change 2.3 ± 2.6, Anger Recall anger change 2.6 ± 2.5) relative to the Stroop Color-Word task (Stroop anger change 1.2 ± 2.1).

Education level was identified as a control variable for the further planned analyses because of its significant correlations with both anger ratings (see Table 8.) and with hostility measures (Total Hostility, $r = -.341$, $p = .009$; Composite Hostility, $r = -.373$, $p = .004$; Cynicism, $r = -.383$, $p = .003$; and Aggressive Responding, $r = -.291$, $p = .028$). No other demographic characteristics or cardiovascular risk factors beyond age or education level were associated in a notable manner with either anger responses or hostility measures. Further, the analysis of peak anger ratings across tasks provided no added information beyond that provided by individual tasks, and will not be examined in further analyses. Peak anger ratings, the highest anger rating among the ratings for each of the three tasks, rather than an aggregate measure of anger implementing the mean across the three tasks, were tested assuming the effects related to anger expression would

be better represented by the peak anger expression response than they would by a mean anger expression measure. In part, this reflects the assumption that both cardiovascular reactivity and myocardial ischemia are related to anger expression in a dose-response manner, such that the effect of anger expression is greatest at its peak levels.

Regression models were employed to identify the predictive value of hostility measures on anger expression following the three tasks. Separate models were examined for Total Hostility, Composite Hostility, and components of Composite Hostility. Predictors of anger expression during tasks in the following regression analyses was assessed by inclusion of the aggregate baseline anger rating in the initial sets of covariates, with the actual post-task anger rating serving as the dependent variable.

Total Hostility

For anger expression following the three tasks, the following relationships with Total Hostility were found. Total Hostility marginally added to a regression model with the covariates of aggregate baseline anger ratings, gender, age, and education in predicting post-Math anger ratings ($R^2 \Delta = .061$, $p = .066$; see Table 9a.). Total Hostility also marginally added to a model with the same covariates in predicting post-Anger Recall anger ratings ($R^2 \Delta = .039$, $p = .095$; see Table 9b.). Total Hostility was not a significant predictor of anger ratings following the Stroop Color-Word stressor ($R^2 \Delta = .035$, $p = .210$; see Table 9c.).

Composite Hostility

For anger expression following the three tasks, the following relationships with Composite Hostility were found. Composite Hostility significantly added to a regression with the covariates of aggregate baseline anger ratings, gender, age, and education in

predicting post-Math anger ratings (R^2 Δ = .081, p = .034; see Table 10a.). Composite Hostility was not a significant predictor of anger reactivity following the Anger Recall stressor (R^2 Δ = .034, p = .118; see Table 10b.) or the Stroop Color-Word stressor (R^2 Δ = .029, p = .254; see Table 10c.).

Composite Hostility components

Multivariate analyses of the components of Composite Hostility are presented in Tables 11a through 11d. These analyses use the three components of Composite Hostility (Cynicism, Aggressive Responding, and Hostile Affect) as predictor variables for each of the three tasks. The approach to determining anger expression was similar to that in the previous two sets of regression analyses, with aggregate baseline anger included in the initial set of covariates, and the post-task anger rating as the dependent or criterion variable. Hostile Affect was a statistically significant predictor for all three of the tasks. Hostile Affect predicted anger expression following the Math stressor (R^2 Δ = .125, p = .008; see Table 11b.) and the Anger Recall stressor (R^2 Δ = .064, p = .031; see Table 11c.). It was a significant predictor of anger expression following the Stroop Color-Word task (R^2 Δ = .162, p = .006; see Table 11d.). Cynicism and Aggressive Responding did not significantly predict anger expression to any of the three tasks.

Hypothesis 1 summary

In summary of the findings regarding the first hypothesis, that hostility measures would be positively related to greater anger expression following the stress tasks, bivariate correlations revealed that Total Hostility was marginally predictive of anger expression to the Math and Anger Recall stressors, but was not predictive of anger response to the Stroop Color-Word task following adjustment for covariates. Composite

Hostility was a significant predictor of anger expression to the Math stressor, and trended towards being predictive of anger reactivity to Anger Recall. The Composite Hostility component of Hostile Affect was a significant predictor of anger expression to all three tasks.

Hypothesis 2.

Hypothesis 2 predicted that hostility would be positively correlated with cardiovascular reactivity measures of systolic and diastolic blood pressure, and heart rate, and further predicted that hostility would be predictive of the presence/absence of mental stress-induced myocardial ischemia. Anger expression was likewise hypothesized to be predictive of both cardiovascular reactivity and ischemia, additive to the contributions of hostility.

Cardiovascular measures and reactivity, hostility, and anger

Correlational analyses were conducted to examine the bivariate relationships between the cardiovascular measures and hostility measures and anger expression (see Table 12a. for relationships with actual cardiovascular measures, see Table 12b. for relationships with change scores of cardiovascular measures). Correlations between cardiovascular measures and hostility measures revealed only one significant relationship. Cynicism was negatively correlated with systolic blood pressure during the math task ($r = -.26$, $p = .048$).

Cardiovascular measures were significantly correlated with anger expression following both the Math and Anger Recall stressors. Anger expression following the Math task was associated with a higher aggregate baseline heart rate and higher heart rate

during Anger Recall (r = .38, *p* = .004; r = .33, *p* = .013, respectively). Anger expression following the Anger Recall task was associated with higher systolic blood pressure, diastolic blood pressure, and heart rate during Anger Recall (r = .30, *p* = .026; r = .36, *p* = .008; r = .30, *p* = .025, respectively).

An index of cardiovascular reactivity was obtained by subtracting mean baseline cardiovascular measures from the mean of the during task measures (simple change scores). For example, the SBP Change for the Math task was the mean of SBP measures during the task minus the mean of the pre-Math SBP measures. Correlations between these simple change scores and hostility measures yielded a number of significant results. Contrary to predictions, Total Hostility was negatively correlated with SBP and DBP change during the Math task (SBP: r = -.41, *p* = .001; and DBP: r = -.27, *p* = .042). Total Hostility also negatively correlated with HR changes during the Stroop Color-Word task (r = -.30, *p* =.043). Composite Hostility was negatively correlated with SBP change during the Math task (r = -.40, *p* = .002). The Composite Hostility components of Cynicism and Hostile Affect were both negatively associated with SBP change during the Math task (Cynicism: r = -.39, *p* = .003; Hostile Affect: r = -.36, *p* = .007). Cynicism was also negatively correlated with DBP changes during the Math task (r = -.28, *p* = .033), and Hostile Affect was negatively correlated with HR changes during the Stroop task (r = -.38, *p* = .010). Thus correlational findings do not support the hypothesis of CVR being positively related to hostility measures, however these effects may be biased by the negative correlation between age and hostility.

The anger-cardiovascular reactivity relationship was also explored by correlational analyses. Anger expression following the Anger Recall task was

significantly positively correlated with SBP, DBP, and HR changes during that task (SBP: r = .36, p = .007; DBP: r = .31, p = .022; HR: r = .32, p = .018). These correlations mirror those found in the absolute Anger Recall cardiovascular measures, and support the hypothesis of anger expression as predictive of cardiovascular reactivity. Anger expression following the Math task was otherwise not notably associated with any CVR measure, nor was anger expression following the Stroop task significantly associated with cardiovascular reactivity measures.

Initial correlations and t-tests were also conducted to assess the relationship between cardiovascular level and reactivity measures and participant demographic and clinical characteristics. Because of its significant relationship with cardiovascular measures, diabetes status was identified as a control variable for the further planned analyses. Diabetes was associated with higher aggregate baseline systolic blood pressure [t(56) = 2.19, p = .032], higher mean systolic blood pressure during the Stroop Color-Word task [t(43) = 2.03, p = .048], and higher aggregate baseline heart rate [t(56) = 2.22, p = .031]. History of prior coronary angioplasty was associated with higher aggregate baseline heart rate, and higher heart rates during the Math and Stroop tasks respectively [t(54) = 2.21, p = .031, t(15) = 2.18, p = .045, t(11) = 3.19, p = .009], but was not included as a covariate because of its low rate of occurrence overall and being associated only with heart rate. Education level was significantly positively associated with DBP levels during the Math task (r = .34, p = .011), and with HR levels during the Math task (r = .28, p = .042). Education level was also significantly associated with the two corresponding reactivity (simple change) measures during the Math task, DBP (r = .44, p = .001), and HR (r = .41, p = .002). Education level was significantly associated

with SBP change during the Math task (r = .39, p = .004) and HR change during the Stroop Color-Word task (r = .36, p = .015). No other demographic or clinical characteristics beyond those mentioned above or those already planned for analysis were associated in a notable manner with either cardiovascular levels during the tasks or cardiovascular reactivity measures.

Regression analyses of cardiovascular reactivity- hostility and anger

Set-wise hierarchical regression analyses were conducted to assess the effects of hostility and anger expression on the three measures of cardiovascular reactivity (systolic and diastolic blood pressure and heart rate). Initially, reactivity was considered by inclusion of the aggregate baseline of the cardiovascular measure within the set of control variables, and the "during task" cardiovascular measure as the criterion or dependent variable. However, due to the high correlations (r's > 0.75) between task and baseline hemodynamics, simple change scores of the cardiovascular measures, rather than the "during task" level measures, were used in the presented analyses as the dependent variables in the following regression models. The calculation of these change scores, noted earlier, was accomplished by subtracting the mean pre-task baseline cardiovascular measure from the mean during-task cardiovascular measure.

Age, gender, education, diabetes, hypertension, number of vessels diseased, and the appropriate aggregate baseline cardiovascular measure were first entered as control variables. Following the set of control variables, the hostility measure was entered (Composite Hostility). The next variable entered was the aggregate baseline for anger expression across the three tasks, followed by the anger expression appropriate to the specific task considered in each separate analyses. Lastly, the hostility measure X anger

expression interaction was entered. This interaction was not statistically significant for any of the cardiovascular measures across the three tasks and will not be further presented in the results.

The findings of the regression models assessing the ability of hostility and anger expression to predict cardiovascular reactivity (CVR) will be presented by task.

For the Math task, systolic blood pressure (SBP) changes were significantly predicted by Composite Hostility after covarying for control variables ($R^2 \Delta$ .082, p = .023, see Table 13a.). Composite Hostility was not a significant predictor of diastolic blood pressure (DBP) or heart rate (HR) changes during the task. Anger expression was not a significant predictor of cardiovascular reactivity during the Math task.

For the Anger Recall task, Composite Hostility was not a significant predictor of CVR. Anger expression, assessed as post-task Anger ratings, covarying for aggregate baseline anger rating, was predictive of cardiovascular reactivity. It was a significant predictor of SBP changes ($R^2 \Delta$ = .126, p = .014; see Table 13b.) and DBP changes ($R^2 \Delta$ = .186, p = .002; see Table 13c.) during Anger Recall after covarying for demographics, risk factors, and Composite Hostility. Lastly, anger ratings from the Anger Recall task showed a marginally significant relationship to heart rate changes during the task ($R^2 \Delta$ = .055, p = .072; see Table 13e.).

For the Stroop Color-Word task, Composite Hostility was a marginally significant predictor in the overall model for DBP changes that also factored in the contribution of anger reactivity (β = 0.334, p = .068, see Table 13d.). Anger expression was also a significant indicator of DBP change during the Stroop Color-Word task ($R^2 \Delta$ = .077, p = .044; see Table 13d.), however, the negative correlation between anger ratings during the

task and DBP change during the task reveal that the anger response was inversely associated with DBP change after controlling for hostility.

In summary of analyses of the relationship of hostility and anger expression with cardiovascular reactivity, there was weak evidence relating Composite Hostility to cardiovascular reactivity. Composite Hostility significantly predicted systolic blood pressure changes during the Math task, and marginally contributed to a model for diastolic blood pressure changes during the Stroop task. However, anger expression was more frequently associated with cardiovascular reactivity, showing significant relationships after covarying for demographics, risk factors, and Composite Hostility in models predicting systolic and diastolic blood pressure reactivity. Anger expression was a marginally significant predictor of heart rate reactivity during the Anger Recall task. Lastly, anger expression was predictive of diastolic blood pressure reactivity during the Stroop task, albeit, in a direction contrary to that hypothesized.

Myocardial Ischemia

Hypothesis 2 also proposed that hostility and anger would be predictive of myocardial ischemia. The relationship between the presence/absence of ischemia during each task and the five hostility measures were explored with cross-tabulation and by chi-square analysis. Total Hostility was dichotomized at the median, with scores of 18 or higher classified as "high." Composite Hostility was split at the median with scores of eleven or greater classified as the "high" group. The median split for Cynicism classified scores of six or greater as "high." High Hostile Affect (determined by median split) was designated for Hostile Affect scores of two or greater, and Aggressive Responding high scores, as determined by median split were those greater than three.

The Total Hostility by Ischemic Status (presence/absence) chi-square analyses (see Tables 14a-c.) were marginally significant for the Anger Recall task ($\chi^2 = 2.94$, $p = .087$) and the Stroop task ($\chi^2 = 3.09$, $p = .079$). Visual inspection of the Anger Recall groups through cross-tabulation (see Table 14b. and Figure 4.) showed greater numbers of the ischemia-positive participants were high in Total Hostility (9 participants) than were low in Total Hostility (5 participants), and the opposite ratio applied for those that did not experience ischemia during the Anger Recall task- 19 non-ischemic participants were low in Total Hostility, whereas only 11 non-ischemic participants were high in Total Hostility. The cross tabulation for presence/absence of ischemia during the Stroop Color-Word task (see Table 14c. and Figure 5.) revealed a similar trend: among the participants that displayed ischemia during the task, 6 were high in Total Hostility, and 3 were low in Total Hostility. Among the non-ischemic participants in the Stroop task, 18 were low in Total Hostility, whereas only 9 were high in Total Hostility. These findings are suggestive of a possible relationship where myocardial ischemia appears more likely for those higher in Total Hostility in the Anger Recall task and Stroop Color-Word tasks. The same results were not obtained in the analysis of Total Hostility by presence/absence of myocardial ischemia during the Math task (Table 14a.). Chi-square tests revealed no relationships between myocardial ischemia and Composite Hostility for the three tasks, Math, Anger Recall, and the Stroop Color-Word task ($\chi^2 = .007$, $p = .932$; $\chi^2 = 1.13$, $p = .287$; $\chi^2 = .952$, $p = .329$, respectively). The three Composite Hostility subcomponents of Cynicism, Hostile Affect, and Aggressive Responding also appeared to be unrelated to ischemic status across the three tasks, with no significant chi-square values from the analyses of the dichotomized component scores and ischemia. Total Hostility and

Composite Hostility by presence/absence of ischemia for any of the three tasks (aggregating across the three tasks) chi-square analyses were also conducted (one for Total Hostility, the other for Composite Hostility). Neither Total Hostility nor Composite Hostility was associated with presence of ischemia overall (Total Hostility: $\chi^2 = 2.581$, $p = .108$; Composite Hostility: $\chi^2 = 1.008$, $p = .315$).

The relationships between anger expression and ischemic status (presence/absence of ischemia) by task were explored with cross-tabulation and by chi-square analysis (See Tables 15a-c.). Anger expression (the difference in anger ratings pre- to post-task, with the actual anger ratings ranging from 1 to 7) following the Math task was dichotomized (median split), with "low anger change" ranging from –3 to 1, and "high anger change" ranging from 2 to 6. The anger ratings following the Anger Recall task were split at the median with low anger change scores ranging from –2 to 2, and high anger change scores from 3 to 6. Lastly, the median split for anger change scores from the Stroop Color-Word task were classified as low for scores from –6 to 0, and high anger change scores ranged from 1 to 6. Chi-square tests revealed no relationships between anger change scores and the presence/absence of myocardial ischemia for the three tasks, Math ($\chi^2 = .046$, $p = .830$; see Table 15a.), Anger Recall ($\chi^2 = 1.123$, $p = .289$; see Table 15b.), and the Stroop Color-Word task ($\chi^2 = .000$, $p = 1.000$; Table 15c.). Ischemia for any task (aggregating across the three tasks) was not significantly associated with the mean of the simple change scores for anger expression ($\chi^2 = .471$, $p = .493$).

Similar to the previous analysis of cardiovascular reactivity, participant demographics and clinical characteristics were inspected for relationships with ischemic status during each of the three tasks. These analyses were conducted through logistic

regressions or chi-square analyses. None of the demographic or clinical characteristics of participants were significantly associated with myocardial ischemia beyond those variables designated a priori for analysis in logistic regressions (age, gender, hypertension, and number of vessels diseased).

Summary

To summarize the chi-square analyses between ischemic status and anger rating changes, anger rating changes appear independent of ischemia. In summary of the chi-square analyses between the presence/absence of myocardial ischemia and hostility measures, Total Hostility was marginally related to the presence of myocardial ischemia during the Anger Recall and Stroop Color-Word tasks.

As noted in the Statistical Analyses section, multivariate logistic regressions were planned following positive findings from the chi-square analyses to account for possible covariates. Despite failure to find significant relationships through chi-square analyses between Composite Hostility and stress-induced myocardial ischemia, or between anger expression and stress-induced myocardial ischemia, logistic regressions were conducted to inspect hypothesized relationships between anger and hostility and myocardial ischemia.

Analyses were conducted for the dependent variable of presence/absence of ischemia related to each of the three tasks. The first set consisted of control variables: age, gender, hypertension, and number of vessels diseased. The second set consisted of Composite Hostility, and the last set was the anger rating change specific to the task, obtained by entering the aggregate baseline anger rating followed by the task-specific anger rating.

The logistic regression examining ischemia during the Math stressor suggested no significant contribution from Composite Hostility (B= -1.68, S.E. = .131, *p* = .846) or from anger expression (B= -.458, S.E. = .412, *p* = .266). The logistic regression examining ischemia during the Anger Recall stressor revealed no significant contribution from Composite Hostility (B= .075, S.E. = .090, *p* = .402) or from anger expression (B= -.279, S.E. = .245, *p* = .255). Lastly, ischemia during the Stroop Color-Word task was not significantly predicted by Composite Hostility (B= .029, S.E. = .102, *p* = .775), nor were anger ratings significantly predictive of ischemia (B= -.422, S.E. = .417, *p* =.312). Thus these analyses revealed no significant relationships between ischemia and either hostility or anger. The lack of power for these analyses may account for these results and is considered further in the Discussion.

Hypothesis 3.

Hypothesis 3 considered the role of defensiveness in addition to and in conjunction with hostility relative to cardiovascular reactivity and myocardial ischemia. It was predicted that defensiveness would be associated with lower reported anger and higher reported interest changes in response to the mental stress tasks. It was further predicted that defensiveness would result in greater cardiovascular reactivity and ischemia. Lastly, it was predicted that defensiveness would mediate the hostility/cardiovascular reactivity relationships, and hostility/ischemia relationships, with greater defensive levels combined with higher hostility leading to greater cardiovascular reactivity and stress-induced myocardial ischemia.

Defensiveness and anger/interest ratings

Pearson product-moment correlations between anger expression levels as well as anger expression changes for each task and defensiveness were statistically non-significant (see Table 16a. for correlations with anger expression levels, Table 16b. for correlations with anger expression changes). Mixed-model analyses of variance for each task were conducted. Anger ratings before and after task were the within-subjects factor with defensiveness (median split) as the between-subjects factor. There were no significant interactions between anger ratings and defensiveness for the Math task, the Anger Recall task, or the Stroop Color-Word Task [$F (1, 50) = .143$, $p = .707$; $F (1, 50) = .856$, $p = .359$; and $F (1, 41) = .677$, $p = .415$, respectively].

Defensiveness and cardiovascular reactivity

Pearson product-moment correlations between defensiveness and cardiovascular reactivity measures revealed non-significant associations, with the exception of a negative correlation between defensiveness and systolic blood pressure change during the Stroop task ($r = -.352$, $p = .019$; see Table 17b.). This correlation's direction is contrary to that predicted by the third hypothesis, which expected greater reactivity with higher levels of defensiveness.

Prior to regression analyses concerning defensiveness and cardiovascular reactivity, correlations and t-tests were conducted between defensiveness and participant demographic and clinical characteristics. These analyses revealed no potential confounding variables beyond those planned or identified from earlier analyses.

Setwise hierarchical regressions were conducted to assess the role of defensiveness in addition to previously considered variables of hostility and anger

expression in eliciting cardiovascular reactivity to the three mental stress tasks. Cardiovascular reactivity was assessed in the same manner as was done with Hypothesis 2: the aggregate baseline of the cardiovascular measure was included in the initial set of covariates, with the dependent or criterion variable being the simple change score of pre- to during-task means of the cardiovascular measure. Change scores were employed as the dependent variable instead of the planned "during task" cardiovascular measures due to high correlations between pre-task and during-task cardiovascular measures. After the first step of individual control variables (age, gender, education, hypertension, number of vessels diseased, and aggregate baseline of the specific blood pressure measure), Composite Hostility was considered as the predictor variable. The third set added defensiveness, and the fourth set considered the Composite Hostility X defensiveness interaction. The fifth set evaluated the contribution of anger reactivity, entering both aggregate baseline ratings and then post-task anger ratings (levels) into the model.

The inclusion of the Composite Hostility X defensiveness interaction in the regression model resulted in an unacceptable level of collinearity indicating an unstable model. The analyses thus present the findings without the Composite Hostility X defensiveness interaction included.

Composite Hostility alone was not a significant predictor of cardiovascular reactivity during any of the three tasks after considering demographics and risk factor covariates.

However, within the same analyses, defensiveness was a significant predictor of systolic blood pressure change for the Stroop Color-Word task ($R^2 \Delta = .349$, $p = .008$; see Table 18b.) and was marginally significant in the overall model assessing diastolic blood

pressure changes during the Stroop task when also considering control variables, Composite Hostility, and anger reactivity (β = -.287, p = .078, see Table 18d.). The direction of these effects, however, are different than those hypothesized, in that lower defensiveness was associated with greater systolic and diastolic blood pressure changes during the Stroop task (see Table 17b.). Defensiveness did not significantly add to other models predicting cardiovascular reactivity across the three tasks.

The last step in the analyses considered anger expression. Anger expression (post task anger ratings after covarying for pre-task aggregate baseline), as in the analyses for Hypothesis 2, were significant predictors of systolic and diastolic blood pressure changes during Anger Recall ($R^2 \Delta$ = .138, p = .010; $R^2 \Delta$ = .154, p = .006; respectively, see Tables 18a and 18c.). Also, as with Hypothesis 2, anger expression during the Stroop Color-Word task was significant in a model to predict diastolic blood pressure ($R^2 \Delta$ = .085, p = .030, see Table 18d.).

Summary

In summary of analyses of the association of defensiveness with hostility and cardiovascular reactivity, defensiveness alone was associated with cardiovascular reactivity in one case significantly, in another, marginally, however, those associations were in a direction contrary to that expected by the third hypothesis. Hostility measures alone also were not strongly related to cardiovascular reactivity. Anger ratings changes, as in Hypothesis 2, were associated with cardiovascular reactivity, most notably with reactivity during the Anger Recall task.

Defensiveness and myocardial ischemia

Defensiveness was dichotomized (median split) in order to perform a chi-square test for independence between defensiveness and ischemic status during the three tasks (see Tables 19a-c). High defensiveness was defined as a Marlowe-Crowne score of greater than 19, with exactly 50 percent of scores in this group. Chi-square analysis between the dichotomized defensiveness groups and myocardial ischemia during the Math task was not statistically significant ($\chi^2 = .655$, $p = .418$). Defensiveness also was unrelated to the presence of stress-induced myocardial ischemia during the Anger Recall task and the Stroop task ($\chi^2 = .006$, $p = .936$; $\chi^2 = .001$, $p = .982$, respectively). Ischemia for any task (aggregating across the three tasks) was not significantly associated with defensiveness ($\chi^2 = .793$, $p = .373$).

As with analyses of stress-induced myocardial ischemia in Hypothesis 2, multivariate logistic regressions were planned following positive findings from the chi-square analyses to account for possible covariates. Despite failure to find significant relationships between defensiveness and stress-induced myocardial ischemia through chi-square analyses, the logistic regressions were conducted to inspect hypothesized relationships between hostility, defensiveness, and myocardial ischemia. Composite Hostility alone was not a significant predictor of myocardial ischemia. Defensiveness alone was not a significant predictor of stress-induced myocardial ischemia. The Composite Hostility X defensiveness interaction, as with the regression models to predict cardiovascular reactivity, resulted in an unacceptably unstable model due to the high correlation between the interaction factor and the individual variables of Composite Hostility and defensiveness (see Table 20.), precluding analysis of their interaction.

Another approach to inspecting an interaction effect was implemented by using the median splits of Composite Hostility and the Marlowe-Crowne Social Desirability Scale to create the Defensive Hostility variable employed in previous studies (e.g. Helmers, et al., 1995). This results in four groups, labeled, in a fashion similar to previous studies, Low Hostile (LH; low Composite Hostility, low Marlowe-Crowne), High Hostile (HH; high hostility, low defensiveness), Defensive (Def; low hostility, high defensiveness), and Defensive Hostile (DH; high hostility, high defensiveness). The number of participants for each group were as follows: 9 were Low Hostile; 13 were High Hostile; 16 were Defensive; and 7 were Defensive Hostile. The percentage of participants that were positive for mental stress-induced ischemia during any of the tasks varied, with 44 percent of Low Hostile participants positive for ischemia, 31 percent for both the High Hostile and Defensive groups positive for ischemia, and 71 percent of the Defensive Hostile group positive for ischemia ($\chi 2 = 3.93$, $p = .269$; see Table 21. & Figure 6.). Although the chi-square is statistically non-significant, visual inspection of Figure 6 is highly suggestive of an interactive effect between hostility and defensiveness.

A logistic regression was conducted with ischemia to any task as the dependent variable, with covariates of age and a dichotomized measure of number of vessels diseased (1 vessel disease compared to 2 and 3 vessel disease). After also covarying for measures of Composite Hostility and defensiveness that were dichotomized at median splits, the interaction term of those two variables was a marginally significant predictor of stress-induced myocardial ischemia, however the confidence intervals for the estimated odds ratio related to the interaction were extremely large [$p = .054$; Exp (B) 41.68, C.I. .939 – 1849.75].

The Defensive Hostility paradigm was also analyzed with respect to measures of cardiovascular reactivity. A series of 2 X 2 X 2 repeated measures analysis of variance designs were analyzed, with 2 level between-subjects classifications of high/low Composite Hostility and high/low Defensiveness and the repeated measures terms of cardiovascular responses (SBP, DBP, and HR) during baseline and the tasks (considered in separate analyses). There were no significant three-way interactions indicative of differential cardiovascular reactivity based on dichotomous splits of Composite Hostility and Defensiveness. Thus in this analysis, Defensive Hostility was not a significant factor in changes in systolic or diastolic blood pressure, or heart rate in any of the three tasks. To consider anger responses in relation to dichotomized defensiveness, a series of 2 X 2 X 2 repeated measures analysis of variance designs were also analyzed in a similar fashion, with dichotomized anger change scores for each task as a between-subjects factor, rather than Composite Hostility, with high/low Defensiveness as the second between-subjects factor, with repeated measures terms of cardiovascular responses pre- and post-task. These analyses also revealed no significant three-way interactions suggesting differential cardiovascular reactivity based on the dichotomous splits of anger responses and defensiveness.

DISCUSSION

This dissertation addressed questions about relationships between trait measures of personality and expression of emotional states. Specifically, trait-like measures of hostility and defensiveness, and expressions of anger and interest before and after mental stress challenges were studied. The second main focus of the dissertation was examining how those personality and emotional factors related to cardiovascular reactivity and myocardial ischemia. The study found that hostility, as assessed by the Cook-Medley Hostility Scale, was significantly associated with anger expression following mental stress challenges. Anger expression, but not hostility or defensiveness, was a significant predictor of cardiovascular reactivity. Mental stress-induced myocardial ischemia was not significantly associated with hostility, anger expression, or defensiveness in analyses that, unfortunately, were underpowered. In sum, the most prominent findings of the study were the relationships between hostility and anger expression, and that anger expression was a significant predictor of cardiovascular reactivity, most notably in the Anger Recall task. The discussion will consider each of these points in turn, first inspecting relationships among hostility, anger, and defensiveness, and then their relationships to cardiovascular reactivity. The findings regarding potential relationships between hostility, anger, and defensiveness with myocardial ischemia will next be discussed. Consideration of limitations of the study and future research directions are then followed by concluding remarks.

Relationships between psychological variables

Hostility and anger expression

Cook-Medley Total Hostility and Composite Hostility mean scores were

comparable to those found in previous studies of both healthy and CAD populations (Barefoot et al., 1989; Helmers et al., 1991; Helmers & Krantz, 1996). Anger reports as obtained in this study are not easily compared to other anger measures, being unique to the specific stressor situations, and employing a Likert-type scale not frequently implemented in studies of anger. Relationships between hostility measures and anger expression were consistently supportive of the first hypothesis, that higher hostility would positively correlate with greater anger expression related to mental stress challenges (see Table 8.). Additionally, hostility measures were positively associated with aggregate baseline anger expression as well. This suggests not only that hostility is associated with greater anger reactivity, but also that it potentially contributes to a more stable underlying trait-like anger as well, or at a minimum, a greater likelihood of identifying and reporting higher levels of anger in the absence of any apparent stimulus. Correlational analyses further indicated significant positive relationships across tasks, such that individuals rating higher anger following one of the tasks was quite likely to also report higher anger states following the other two tasks. Thus, although there were differences in anger expression between tasks, the correlations of anger expression between tasks suggests that there are shared qualities among the tasks which contribute to the anger responses.

The regression analyses employing hostility measures to predict anger expression to each of the tasks generally confirmed the correlational findings. Total Hostility marginally predicted anger expression to the Math stressor and the Anger Recall stressor. Composite Hostility in a similar manner was a significant predictor of anger expression following the Math stressor, and was marginally predictive of anger expression following the Anger Recall Stressor. Of the three Composite Hostility components, Hostile Affect

contributed the most to models predicting anger expression, with significant increases in overall variance accounted for by models related to all three tasks. Thus, while there did not appear to be a considerable difference between Total versus Composite Hostility, the Composite Hostility component of Hostile Affect contributed the greatest increases in predictive value in models for anger expression across the three tasks. The label applied to the Composite Hostility component is particularly relevant in comparison to the other two components of Cynicism and Aggressive Responding. Hostile Affect implies that the hostility is communicated or manifest in an affective response, which would appropriately be manifest by anger expression such as the anger reports provided in relation to the mental stress tasks. In answering how angry one *feels* following a task, affect would seem to play a larger role than would cynicism (an attitude) or aggressive responding (a behavioral indicator).

In examining the hostility/anger expression relationship, it is interesting to consider the factor of education level. Education level was a factor positively correlating with anger reports post-task, while being negatively correlated with aggregate baseline anger and each of the hostility measures. That would suggest a number of possibilities, one being that education level provides some indication of an individual's sensitivity to their attitudes and emotions-- greater education equipped individuals with a greater awareness and willingness to report the experience of anger as well as contributing to lower anger and hostility in the absence of threat. Another possibility is that greater education provides participants with more sensitivity to the appropriateness of their responses- in essence, that they recognize when they should and should not report anger, irrespective of the actual experience, a variation on the "good subject" problem.

Although hostility measures, as hypothesized, predicted increased anger expression during mental stress tasks after accounting for baseline values and control variables, none of the models predicted much greater than one third of the variance in predicting anger expression during each of the tasks. This suggests that other factors not considered by this research can provide important information in understanding anger experiences in stressful situations. Such factors may include measures of trait anger, as in previous studies where anger expression and suppression are associated with hostility measures in correlations varying in strength (Smith & Frohm, 1985; Hardy & Smith, 1988; Suarez & Williams, 1990; Helmers, 1993). These studies differ from the dissertation in the way they measured anger. These studies related hostility to questionnaire measures of anger such as Speilberger's scales of Anger-in and Anger-out (1985), whereas the dissertation employs the Likert rating tied specifically to a task and its preceding baseline period. A combination of both state and trait anger assessment may yield a more predictive model.

Other factors that might improve the predictive value of the model may include consideration of more general measures of positive and negative affect and dispositional expressivity (e.g. Gross, John, & Richards, 2000). Gross and colleagues reported that emotion expression depends not just on an individual's disposition to be highly emotionally expressive or to withhold emotion expression, but also on the nature of the emotion. In their studies, experience was related to emotional expression only for dispositionally high-expressivity participants, but not for low expressivity participants when pertaining to negative emotions, but the two groups did not differ in emotional expressivity related to positive emotions. Finally, behavioral measures of anger may be

considered in an effort to further clarify the hostility-anger relationship.

Anger expression, interest, and defensiveness

Defensiveness scores obtained by scoring of the Marlowe-Crowne Social Desirability Scale were comparable to those obtained in previous studies (Mann & James, 1998; Helmers et al., 1995). Defensiveness was predicted to be negatively associated with anger expression and positively associated with expression of interest following the three mental stress tasks. While the correlations between anger expression and defensiveness were statistically non-significant and of small magnitude, they were in the predicted direction. Mixed model analyses of variance also revealed no significant interactions between anger expression (pre- and post- as the within subjects factor) and defensiveness (between subjects factor). Given those findings, the hypothesized negative relationship between anger expression and defensiveness was not supported.

The correlations between defensiveness and expression of interest were positive (in the predicted direction) for the Math task, and the Anger Recall task, but negative for the Stroop task. These correlations were also statistically insignificant and of small magnitude. The interest ratings for each of the tasks and their baselines (see Table 5.) showed little change pre- vs. post- task, or across tasks, with a range of less than one point on the seven point scale across all three baseline and three post-task measures. These findings also do not support the dissertation hypotheses.

The failure to find significant relationships between defensiveness scores and anger expression cannot be attributed to the same restriction of range noted in the interest scores, and this study does not demonstrate a significant relationship between defensiveness and expression of either anger or interest following the three mental stress

tasks. While previous work has found defensive subjects express decreased trait anger and score lower on measures of Anger-out (Helmers, 1993), it has been suggested that the Marlowe-Crowne Social Desirability scale measures two constructs labeled self-deception and deception of others (Paulhus, 1984). Helmers (1993) suggested that, in a healthy younger population, men and women differ on the trait of "other deception" (based on reference to a third measure), and that analyses considering defensiveness should therefore evaluate men and women separately. This dissertation included only seven women in the total sample of 59, and it is not clear how the role of gender impacts the issue of defensiveness, nor is the differentiation of self- versus other-deception considered within this sample.

While not a component of any of the study hypotheses, this study found that defensiveness was significantly negatively correlated with both Total Hostility and Composite Hostility. This finding is of importance in later considerations of psychological factors related to cardiovascular reactivity, in which the issue of collinearity prevented adequate inspection of the hostility X defensiveness interaction.

To conclude the discussion of the consideration of defensiveness in relation to the expression of anger and interest, it is the author's belief that the lack of the hypothesized negative relationship between defensiveness and anger expression reflects two factors: on the one hand, the tasks' abilities to elicit anger responses, and on the other the inability of the tasks to sufficiently elicit defensiveness. The conditions provided by the tasks, while containing 'evaluative threat' elements such as having an audience during the Math task, and being recorded during the Anger Recall task, were not perhaps threatening enough to engage defensive thoughts or behaviors in the participants. The lack of

defensiveness effects in relation to anger expression may also tie in to the expectations of the participants. First, the participant had no background or previous experience with the researchers, and little likelihood of frequent contact with the researchers following the protocol, reducing the importance of positive self-representation as the participant would suffer few (if any) negative consequences for an other than positive self-representation. Second, the experience within the laboratory protocol, while intended to mimic the stressful circumstances of daily life, may have been too unlike daily life experiences. Participants provided informed consent recognize they are fairly protected from harm during the protocol, to include the expectation that they will not be severely criticized or otherwise subject to the type of experience that might be most threatening to highly defensive individuals.

Psychological variables and cardiovascular reactivity

Hostility and cardiovascular reactivity

Hostility was hypothesized to be positively correlated with increased cardiovascular reactivity, with anger expression mediating the effects of hostility on cardiovascular reactivity. Analyses revealed significant negative correlations between the two hostility measures and changes in systolic blood pressure related to the Math task. This finding that lower hostility scores were associated with greater changes in systolic blood pressure is, however, contrary to hypothesized relationships. Composite Hostility was a marginally significant factor in accord with hypotheses in the regression model predicting diastolic blood pressure changes during the Stroop Color-Word task, but the magnitude of the effect associated with Composite Hostility in the overall model, while statistically significant, was not very large. Other than those two findings

regarding SBP changes during the Math task and DBP changes during the Stroop task, Composite Hostility did not contribute significantly to models for predicting cardiovascular reactivity.

Possible explanations to account for the failure to find the expected increased cardiovascular reactivity related to hostility may center around a type of "inoculation effect" provided by hostile attitudes. Perhaps hostile individuals, with cynicism coloring their perception, expect the mental stress tasks to be unpleasant. Finding no discrepancy between their expectations and the actual state of affairs, they might not have experienced the sympathetic arousal that would trigger changes in blood pressure and heart rate. It is possible that less hostile individuals were likewise taken aback at the nature of the tasks and were more concerned about the unfolding of events in the laboratory, as their perspective is not to expect the researchers to challenge them, or delve into and relive past anger episodes.

Other possible explanations for the failure to find the expected increased cardiovascular reactivity associated with hostility are that the task manipulations were either not powerful enough to induce changes, or that the nature of the tasks elicited sufficiently large changes, but that hostility was not a key factor in inducing the cardiovascular reactivity. Blood pressure and heart rate measures taken before and during the mental stress tasks indicate a clear and statistically significant effect from the task manipulations. It would thus seem unlikely that the lack of a hostility/CVR relationship was due to the tasks lacking the ability to elicit sufficient cardiovascular reactivity generally. It appears that, at least in this study, hostility alone was not a substantial factor in cardiovascular reactivity. A factor that did contribute more

consistently to the models predicting cardiovascular reactivity was that of anger expression related to the tasks, discussed in the following section.

Anger expression and cardiovascular reactivity

Anger expression, significantly higher following tasks compared to baseline measures, was positively correlated with cardiovascular reactivity (CVR) most strongly in the Anger Recall condition, with moderate to large significant correlations to SBP, DBP, and HR following Anger Recall. Not surprisingly, regressions considering anger expression following the Anger Recall task revealed relationships of moderate magnitude, with anger expression significantly predictive of SBP and DBP changes to the task, and marginally predictive of HR changes.

This relationship between anger expression and cardiovascular reactivity during the Anger Recall task may be interpreted as an indication of how anger states may impact the cardiovascular system in stressful situations. The Anger Recall task requires the participant to recount a recent incident during which she became angry, irritated, frustrated or upset. The experimenter guides this recounting to focus on the participant's emotions- when she is most upset and angry. The type of anger this task elicits focuses on personally relevant anger, and the task also requires a level of consciousness and reflection which would likely involve the frontal lobe and cortical influences. One of the assumptions of the experimenter in this case is that the participant will not only recall that previous experience, but to a degree, experience those emotions yet again, and that this re-experiencing of the emotions leads to the sympathetic response indicated by cardiovascular reactivity. The fact that anger expression is positively related to cardiovascular reactivity during the task would suggest a validation of this assumption.

Anger expression following the Math task did not significantly correlate with cardiovascular reactivity during the task. Regression analyses considering the role of anger expression in predicting cardiovascular reactivity likewise did not reveal a significant contribution of anger expression. The failure to find the same relationships during this task as were found in the Anger Recall task may possibly be explained by the nature of the tasks. In the Math task, the participant's focus is more internal, and he often expresses frustration or irritation with himself regarding his performance. The experimenter provides interference during the task, interrupting, correcting, and otherwise verbally pushing the participant, but it is not uncommon for the participant to comment later that the experimenter was "just doing his job." There seemed, at an anecdotal level, to be a reluctance to direct negative emotions towards the experimenter. The task did, however, elicit anger reports comparable to those elicited by the Anger Recall task. The math task involves the interpersonal interactions between experimenter and the participant, but the anger response has less of a reflective element than in the Anger Recall task. It is quite possible that this anger develops largely at the subcortical level, as the participant is put "on the spot" and is not permitted to let her thoughts stray from the mental arithmetic task. Its expression is exclusively in the rating provided after the task. This is in contrast to the Anger Recall situation, where the experimenter wants to hear about the participant's anger with another individual, and encourages the participant to relive that anger. The participant is "given permission" to be angry, and the anger is also acceptable in that it is not directed at the experimenter, but to a third party not present during the task.

The Stroop Color-Word task is another task that may more readily be interpreted

as a frustration task than an anger-eliciting task. In indicating responses to the computer, the participant receives feedback in the form of tones from the computer indicating incorrect responses. Further, the pacing changes based on the participant's successes or failures. Comments made by participants following this task more frequently center around their frustrations with themselves that they were responding incorrectly or too slowly. Less often, participants would focus on, or blame, the computer or task as too difficult. The experimenter was uninvolved in the task with the exception of getting the task set up to start, and thus was not an appropriate or available target for anger or frustration. Anger expression following the Stroop Color-Word task, while significantly elevated compared to baseline levels, was of a considerably smaller magnitude relative to anger expression increases following the other two tasks. The task perhaps elicited a more exogenous type of anger, influenced by subcortical processes, which would in part explain the lower anger expression for the task relative to the others. It is also quite likely that the cardiovascular responses to the task reflect not so much an anger response, but a more general stress response due to the challenges of the task.

Given these characteristics of the Stroop Color-Word task, the significant increase in predictive value of anger expression in a regression model for DBP changes during the task is not easily explained. The negative correlation between anger response to the Stroop task and DBP change during the task in conjunction with the model indicate an inverse relationship between anger expression and DBP change. It is possible that the decrease in anger is accompanied by increases in some other emotion-related variable, for example feelings of hopelessness or desperation.

Anger expression as a mediator of the effects of hostility on cardiovascular reactivity

It was hypothesized that anger expression related to the three mental stress tasks would serve as a mediator of the effect of hostility on cardiovascular reactivity, with increased anger expression adding to the hostility-cardiovascular reactivity relationship. Interaction effects between hostility measures and anger expression were not significantly associated with cardiovascular reactivity. Further, in the one instance where Composite Hostility was a significant predictor of cardiovascular reactivity, that of SBP change during the Math task, lower hostility was associated with greater change. Anger expression did not significantly add to the overall model. In no other cases did accounting for anger expression provide a significant improvement in the predictive value of Composite Hostility. Thus in this study, anger expression provided predictive value in models for cardiovascular reactivity most effectively in the Anger Recall condition, however, anger expression did not serve as mediators of a hostility-CVR relationship in an interactive manner.

The failure to find the hypothesized mediating effect of anger expression on the hostility/CVR relationship is a direct result of the failure to demonstrate the importance of hostility in predicting cardiovascular reactivity, addressed in a previous section.

Defensiveness and cardiovascular reactivity

Defensiveness was not correlated as predicted with cardiovascular reactivity. When considering earlier hypotheses regarding the relationship between anger expression and defensiveness, it was noted that while those correlations were in the predicted negative direction, they were of small magnitude and statistically insignificant. That finding is reinforced by the regression models revealing anger expression continued to

significantly predict SBP and DBP changes to Anger Recall after consideration of defensiveness. There was no apparent negative relationship between anger reactivity and defensiveness.

The effect of defensiveness was expected to be due to the extra "mental effort" or increased burden of positive self-presentation under challenging circumstances. The analyses of relationships between defensiveness and cardiovascular reactivity, however, provide no evidence of increased effort or stress associated with defensiveness alone. In examining the three tasks, there are tangible efforts to increase evaluative threat in both the Math task and the Anger Recall condition. During the Math task, the participant is told his responses are being reported and scored, and he receives verbal feedback during the task regarding his incorrect responses and insufficient effort. The Anger Recall task is presented as a speech task, to be evaluated and taped by the experimenter, and the white-coated laboratory staff serves as an audience during the task. The Stroop task provides its own automated feedback in the form of computer tones for incorrect responses, which those in the laboratory room may hear, but the researchers are not directly involved in the task. Two explanations for the failure to find a relationship between defensiveness and CVR include the possibility that the evaluative threat manipulations noted above are powerful enough to induce the threat regardless of defensiveness status, or the converse, that there was insufficient evaluative threat activation for those either high or low in defensiveness (a possibility presented when considering the lack of the predicted negative correlation between anger reports and defensiveness). Given the lack of relationship of defensiveness with anger reports in conjunction with the lack of a defensiveness/CVR relationship, it appears the latter is

most likely- that there was insufficient evaluative threat activation.

It is possible, in view of prior research, that defensiveness must be considered in conjunction with hostility (Jamner et al., 1991; Helmers et al., 1995). In the case of this dissertation, then, the impact of defensiveness is not apparent, as hostility traits do not appear to be an important factor in CVR responses to the mental stress tasks. Having to this point discussed the relationships between psychological variables and cardiovascular reactivity, the discussion now addresses the relationships between psychological variables and the dependent variable of myocardial ischemia.

Psychological variables and myocardial ischemia

Hostility, anger expression, and defensiveness were examined for their potential role in elicitation of myocardial ischemia during the three mental stress tasks. Chi-square analyses and logistic regressions were conducted to reveal any potential relationships. In reviewing these analyses, it is important to note a few issues, one being that the number of participants for whom usable data was available is lower than had been anticipated. Given that, there are fewer ischemia cases than anticipated in response to the tasks. While that did not appear to affect results in some cases, there are some analyses that would be expected to reach statistical significance with greater numbers of subjects and ischemic episodes.

Hostility and myocardial ischemia

Chi-square analyses for Total Hostility and stress-induced ischemia revealed trends toward significance for both the Anger Recall task and the Stroop Color-Word task which suggested that participants that were ischemic in response to the mental stress tasks were more likely to be high in hostility, and that non-ischemic participants were

more likely to be low in hostility. This is one set of analyses where larger numbers of participants and cases in the same pattern of findings as with the current data would reveal statistically significant relationships. In interpreting these analyses, the immediate question arises of why the hostility/ischemia relationship, if there, does not manifest itself also in the Math task. The answer is not readily apparent, but one response is considered at this point. It is possible, as noted earlier when considering cardiovascular reactivity, that there are qualitative differences between the tasks that relate to hostility. Assuming that possibility, the Math task might not provide the necessary environment to elicit the ischemia manifest in the other two tasks. To counter that argument, the Math task did result in the lowest rate of stress-induced ischemia relative to the other two tasks, but in absolute terms, the number of ischemic events was the same for the Math and Stroop tasks. Thus, the task elicits myocardial ischemia, but the hostility difference remains unexplained.

Returning to consideration of differences in the nature of the tasks, the Math task would appear to be the one in which hostility might be most readily exhibited, as the participant is corrected and interrupted during the task of counting backwards from a four-digit number. The 'inoculation effect' referred to earlier when considering cardiovascular reactivity might also apply in this circumstance, such that the Math task fits a hostile individual's perception of a proper mental stress challenge. The question then arises of what other, if any, personality or affective element contributed to the ischemia that did occur during the Math task. In the cardiovascular reactivity data, the Math task resulted in the greatest absolute pre- to during-task changes in both systolic and diastolic blood pressure, as well as the greatest changes in heart rate, and it elicits

relatively comparable rates of ischemia as the other tasks, so it clearly results in some physiological effect. It is possible that a better indicator for both cardiovascular reactivity and myocardial ischemia during the mental stress tasks would have been, as noted earlier, some general negative personality or affective measure (which might lead to a non-specific sympathetic arousal) rather than the specific hostility or anger measures employed in this study (Gross et al., 2000).

A caution against overinterpreting the suggestive findings of hostility related to ischemia is that these bivariate relationships were not evident in the logistic regression analyses. It is not readily apparent whether this indicates a lack of power associated with low numbers of participants and cases, or if the covariates (such as number of vessels diseased as an index for disease severity) most fully account for the differences suggested by chi-square analysis. Were sufficient numbers of participants acquired with the same patterns of hostility and myocardial ischemia as manifest in the sample obtained, hostility would clearly have played a role in stress-induced myocardial ischemia, which suggests a lack of power. Other possible explanations of the non-significant logistic regression analyses include the possibility that hostility does not relate to stress-induced myocardial ischemia and /or that other personality and affective characteristics would more accurately predict ischemic events in response to mental stress.

In summary of the findings regarding hostility and myocardial ischemia, chi-square analyses were suggestive of a positive relationship, however, multivariate logistic regressions failed to support those suggestive findings. The trends of the hostility-ischemia relationship for the Anger-Recall and Stroop Color-Word task fit the conceptual model as tested in the hypotheses, suggesting that greater hostility is associated with

greater ischemia. Larger samples providing the ability to adequately inspect the relationship in multivariate models should be obtained to validate or refute those marginally significant findings.

Anger expression and myocardial ischemia

Chi-square analyses and multivariate logistic regressions for anger expression changes predicting ischemia in response to the three mental stress tasks provided no indication of a significant interaction, contrary to predictions. Further, given the proportions indicated in the cross-tabulations, it would not be expected that larger numbers of participants and cases would indicate a positive relationship between anger expression status and ischemic status. In the case of the Anger Recall task, in fact, larger numbers of the same proportions would actually seem to indicate high anger expression during Anger Recall would be protective. As before when discussing hostility in logistic regressions to predict ischemic status, it is not readily apparent whether the failure to find an effect for anger expression during the tasks (alone, or in interaction with hostility) on ischemia indicates a lack of power associated with low numbers of participants and cases, or if the covariates (such as number of vessels diseased as an index for disease severity) most fully account for the differences suggested by chi-square analysis. In considering why in the Anger Recall task, high anger expression might be protective, the author's initial inclination is to return to a suppression/repression explanation. It may be argued that the "high anger expression" group represents a group that does not suppress or repress their anger, whereas the "low anger expression" group represents those that suppress or repress anger. The difficulty with that contention is the lack of relationship found between ischemia and defensiveness, which in this study was considered as a type

of repression or suppression. It is quite possible that the defensiveness measure does not fully capture the type of suppression or repression that comes into play in these cases, and that some other suppression measure would help differentiate participants in conjunction with consideration of anger expression.

It is interesting to note the seeming discrepancy between the cardiovascular reactivity findings and the myocardial ischemia data pertaining to anger and hostility. Anger expression was a significant predictor in multivariate models to predict cardiovascular reactivity measures, but was not significantly associated with ischemic status. Hostility seemed to contribute little in the CVR analyses, but was marginally associated with ischemic status in the Anger Recall and Stroop tasks in bivariate analyses. One possibility in interpreting these findings is to conservatively present that anger expression was related to cardiovascular reactivity measures, but that neither hostility nor anger expression were significantly associated with stress-induced myocardial ischemia. With that interpretation, there is no discrepancy in terms of anger expression predicting CVR, with hostility predicting ischemia, but then the question is why anger expression would be associated with CVR but not ischemia. It is possible that anger expression provides increased risk for ischemia as well as for cardiovascular reactivity, but that the levels of anger expression elicited in the tasks did not cross some threshold level. From another perspective, perhaps the model relating the contributions of hostility and anger expression to CVR are best represented by linear relationships, whereas the risk associated with hostility and anger expression contributing to myocardial ischemia might be best represented by a threshold either not achieved or detected in the current study.

The relationship between CVR and myocardial ischemia also bears further consideration. Cardiovascular reactivity has been implicated in the development of coronary atherosclerosis and/or it may serve as a marker of pathogenic processes. Myocardial ischemia is used clinically both as a marker for or sign of the presence of coronary artery disease, and also is interpreted as contributing to the disease process. Previous research has shown that CAD patients evidencing severe ischemia via radionuclide ventriculography during mental stress also exhibit greater systolic blood pressure increases to mental stress tasks compared to mild-moderate ischemics and healthy controls (Krantz et al., 1991). These researchers suggested that their findings indicate severe ischemia is most likely among CAD individuals with the greatest stress blood pressure reactivity. In discussing the process of developing myocardial ischemia in response to the mental stress tasks, the researchers presented the potential contributions of cardiovascular reactivity in impacting the imbalance of supply and demand of oxygen to the myocardial tissue. Thus, in that study, CVR and ischemia are directly related, with the interpretation that CVR contributes to more severe ischemia.

In contrast, a more recent study reported that CAD patients and healthy controls did not significantly differ in cardiovascular reactivity measures in response to mental stress challenges (Benight et al., 1997). This study, however, contrasted with other studies (Boltwood et al., 1993; Ironson et al., 1992) in that it failed to find decreased perfusion in CAD patients. The sample size of only 6 CHD patients and 9 healthy controls is considerably smaller than these other studies as well. Given the failure to find decreased perfusion in CAD patients, or differential CVR in addition to the small sample sizes it may be questioned whether this study is adequately powered to test differences

between the groups.

For the dissertation study, repeated measures 2 X 2 analyses of variance were conducted to inspect the relationship between cardiovascular reactivity and myocardial ischemia in the current study. The between-subjects classifications were for presence/absence of myocardial ischemia and the repeated measures terms consisted of cardiovascular responses (SBP, DBP, and HR- aggregate baseline and during task measure). The interactions of CV measures by ischemia status for each task were not statistically significant, but there was a main effect for cardiovascular reactivity, meaning simply that there were significant differences comparing CV measures before tasks to during tasks. Thus, there were not significant differences between the ischemia positive and the ischemia negative groups regarding cardiovascular reactivity (see Tables 22a-c for descriptive data). Inspection of means of the actual measures revealed a slightly lower SBP in the ischemia positive group during the three tasks, with almost identical DBP and HR between ischemia negative and positive groups across tasks.

In this dissertation study, the few statistically significant differences regarding medication status related to CVR were all related to heart rate, and the actual absolute differences in heart rate reactivity were fairly small. Ischemia differences by medication status were not significant with the exception of suggestive findings regarding nitrates status during the Math task, with more frequent ischemia in the nitrates withheld group, and nitrates status during the Stroop task, where withholding was also associated with more frequent myocardial ischemia. Overall, the possible effects of medications on cardiovascular reactivity appear fairly minimal. It is possible that withholding of nitrates impacted findings related to ischemia during the Math and Stroop Color-Word task.

Returning to the models presented in the Introduction, the psychophysiological reactivity model (Williams, Barefoot, & Shekelle, 1985) would seem to fit an explanation such as one considered earlier where heightened sympathetic reactivity is thought to contribute to elevated CAD risk. The psychophysiological reactivity model suggests that frequent anger episodes produce elevated levels of cardiovascular and neuroendocrine responses which then contribute to CAD. Anger expression to the Anger Recall task fit this model, as greater anger expression was predictive of greater cardiovascular reactivity. The constitutional vulnerability model (Krantz & Durel, 1983) suggests that basic biological differences cause and explain the psychological and behavioral manifestations of anger, hostility, and aggression. The same underlying biological factor(s), possibly including a hyperresponsive sympathetic nervous system, also confers vulnerability to cardiovascular disease. In the context of this study, CVR and myocardial ischemia could both be interpreted as manifestations of an underlying biological factor that also contributes to attitude, behavioral, and affective differences such as those considered here, hostility, anger expression, or defensiveness. Although the design of the dissertation study was not explicitly constructed to compare these models, the psychophysiological reactivity and constitutional vulnerability models, relative to the others considered in the Introduction, seem to best explain the findings from study.

Defensiveness and myocardial ischemia

Defensiveness was not associated with ischemic status as analyzed by chi-square analysis in any of the three tasks, however, closer examination of the analysis of ischemic status during the Math task suggests a potential relationship between defensiveness and ischemia, with two-thirds of the ischemia-positive group being high in defensiveness (see

Table 19a.). Caution is warranted in assuming that the trend would continue with a larger number of ischemic cases, however, as the total number of ischemic cases during the Math stressor is fairly low. Multivariate logistic regression considerations of the role of defensiveness in ischemia also found no elevated risk for ischemia associated with defensiveness alone. Thus there are no apparent effects of defensiveness on ischemia in bivariate or multivariate analyses.

The alternate approach presented at the end of the Results section, however, was suggestive of an interactive effect between defensiveness and hostility when both variables are dichotomized. The failure to achieve statistical significance in those analyses may be due in large part to the lack of power resulting from lower than expected numbers of participants with available ischemia data. One of the side findings related to the Defensive Hostility approach is that the hostility levels of High Hostile participants were similar to the hostility levels among the Defensive Hostile participants. Some researchers have suggested the possibility that Defensive Hostile individuals are simply "super hostile," but that does not appear to be the case in these findings.

The potential explanations for these findings are similar to those presented in discussion of defensiveness related to CVR, with the assumption, as just discussed, that the mechanisms leading to manifestations of ischemia may be the same as those which result in CVR. It may be that there is no effect of defensiveness on ischemia, or alternatively that the nature of the tasks prevented the demonstration of defensiveness effects on ischemia. There may be a relationship that was obscured by lack of sufficient cases or other undetermined methodological issues.

Study Limitations and Future Directions

In interpreting the results of this study, a number of issues have been presented to this point that may limit the conclusions drawn from the study. One of these is the issue of the study population. The rationale for the study to examine coronary artery disease patients rather than healthy individuals was to take advantage of the unique opportunity to investigate both cardiovascular reactivity and myocardial ischemia. Most studies inspecting psychosocial correlates of cardiovascular reactivity do so with younger, healthier samples, and do not consider stress-induced myocardial ischemia, which is only rarely manifest among healthy persons. Thus, this study is more specific in focusing on psychosocial correlates among CAD patients.

The possibility exists that the findings do not transfer from healthy populations to CAD populations and vice versa, due to the effects of the disease and/or medications. For example, decreased heart rate variability is associated with an increased risk for coronary events and with other cardiovascular risk factors (Colhoun et al., 2001.). Heart rate variability was not assessed in the study group, however, it is quite likely that it may have impacted the results of the study to a greater degree than it would impact the findings from studies with healthy samples. Medication status also may have contributed to differential results, with some associations between heart rate reactivity and use of calcium channel blockers and nitrates, but the overall effects on CVR for medications appear fairly minimal. Myocardial ischemia in this study did not appear to be greatly impacted by medication status with the exception of nitrates withholding being associated with presence of ischemia during the Stroop task. The design of the study, however, involved an effort to mitigate the effects of medications by withholding beta-blockers,

calcium channel blockers and nitrates, and thus the number of participants for whom the medications were prescribed but not withheld was fairly small. Typically, members of this group were not permitted by physicians to withhold their medication for the study due to perceived risks related to disease severity, an element considered within the multivariate analyses. Thus, while use cardiac medications may have attenuated both cardiovascular reactivity and stress-induced myocardial ischemia, the size of that attenuation affect should have been minimal. These complexities related to medicine effects and disease severity are not introduced in other studies that focus exclusively on healthy participants. Such studies, when conducted with long term follow-ups, also allow analysis of risk for development of CAD, whereas this study focuses on risk related to CVR and myocardial ischemia in a population with pre-existing cardiovascular disease.

Researchers have also suggested that there is an effect of labeling, or knowledge of health status on personality measures. For example, Irvine, Garner, Olmsted, and Logan reported that hypertensive patients with knowledge of their hypertensive status scored higher than unaware hypertensives on measures of neuroticism, trait and state anxiety, and self-reported Type A behavior (1989). Aware hypertensives also scored higher on state anger, with a similar trend for anger suppression. The argument presented by these researchers is that these personality factors aren't characteristics of the disease, but reflect the influence of exposure to medical attention or knowledge of hypertension status. It is possible that there were also effects related to knowledge of CAD status in the dissertation study, such that participants' questionnaire measures or anger expression measures may have been higher or lower than they would have been prior to their knowledge of disease status, however, the design of the study prevents quantification of

either the direction or magnitude of such an effect.

Gender

Another related study population issue is gender. Only seven of 59 participants were female, which precluded any analysis of comparisons between males and females. Further, none of the seven females were determined to be ischemic as assessed by radionuclide ventriculography. Previous studies, such as those discussed in the Introduction, have suggested that the psychological factors that may contribute to CVR and ischemia may differ by gender, such as in the likelihood of one gender or the other more frequently possessing or exhibiting a trait or characteristic. Others have also suggested that females and males respond differentially to the three mental stress challenges. Thus, it would be desirable to obtain a similarly large sample of females to allow analyses of the differences by gender.

Possible effects of extending analyses of an existing study

Another limitation of this study pertains to the fact that the study was based on further analyses within data from an existing study, and as such, the larger study was not originally designed for the purposes of the dissertation hypotheses. This leads to a number of possible improvements in study design were the study to be conducted again for validation or other purposes. First, there is the issue of the measures of psychological variables. Other assessments of anger and anger expression could have been implemented to either validate the anger reports, or provide comparison points. Likewise, other approaches to assessing hostility and defensiveness could be implemented to determine which measures most appropriately describe the constructs, and which ones indicate stronger associations with other psychological variables as well

as the dependent variables of cardiovascular reactivity and ischemia.

Second, others have presented findings regarding the importance not only of assessment of psychological variables, but also of having relatively standardized methods of eliciting various affective responses (e.g. Gross, Sutton, & Ketelaar, 2000). The Anger Recall task was the only one of the three mental stress tasks that seemed to tap directly into anger experiences, and the strength of the manipulation depended to a great degree on the nature of the recalled anger event as well as the participant's willingness and ability to revisit that event. Thus, improvements in the specificity and standardization of the tasks pertaining to tapping into hostility, anger, and defensiveness might yield more productive results.

The mental stress manipulations for the dissertation were implemented in the larger TOMIS study to demonstrate environmental influences on cardiovascular stress responding. The intent in studies of environmental influences is for the manipulations to be as strong as possible. This approach may have limited efforts within the dissertation study to identify individual differences, as it is possible the strength of the manipulation washed out, or otherwise obscured the variation that might have been detected were participants exposed to graded levels of the stress-inducing manipulations. Thus modifications to the tasks might also involve providing varying intensity levels of the stressors, in an attempt to more clearly expose individual differences.

A last point related to extending analyses of an existing study is the issue of missing data. The lower numbers of radionuclide ventriculography assessments compared to the cardiovascular reactivity data limited the ability to interpret the data. Cases were excluded when the data was uninterpretable or unavailable. These exclusions

may be attributed to a number of factors, including poor imaging quality and logistical disconnects across institutions over time. Improvements are possible in the methods of data collection and initial analyses, especially in the case of the radionuclide ventriculography data. That is a problem impossible to address and rectify retrospectively, but the problem likely could have been minimized, if not resolved, when addressed in process.

Perhaps more critical to these analyses is the finding that approximately 75 percent of the ischemia-positive reports derived from a laboratory that analyzed only 43 percent of the reports (Cedars-Sinai). It is not clear at this point what may account for the differences in analyses across the laboratories. All three laboratories were provided the same initial data for analysis, with the same instructions and score sheets to report their findings. The participants across laboratories of analysis did not differ regarding scores on measures of hostility, anger expression, or defensiveness, but the participants whose data were analyzed at Cedars-Sinai had a lower mean education level. This difference was considered in analyses, with education level covaried in analyses for each of the three hypotheses. There were no substantial differences between the groups in other patient demographic or clinical characteristics and risk factors, or in their cardiovascular reactivity measures. Thus reliability of RNVG analyses across laboratories was fairly low, with no clear differences between the samples of the laboratories beyond the education level variable to account for those differences.

Future directions

Promising approaches for future research include efforts at replicating the finding that hostility predicts anger expression, and determining under what circumstances the

concordance between trait measures and anger expression is the highest. In conjunction with that finding, the relationship between anger expression and cardiovascular reactivity in the Anger Recall condition contributes to the literature that exists, both in the research and lay public communities, suggesting that the experience of anger is associated with increased sympathetic responding. Further clarification of the mechanisms of that relationship would enhance research and clinical efforts already underway in the domains of anger management, particularly in the case of those at high risk of or those diagnosed with coronary artery disease. Such studies regarding mechanisms could monitor general sympathetic responding as well as cardiovascular responding, and also extend those measures to include considerations of parasympathetic or vagal influences, which some have suggested play an important role (Brosschot & Thayer, 1998).

One implication of the study relates to the finding just noted on the impact of anger expression on cardiovascular reactivity. After controlling for risk factors and hostility, the effect of increased anger expression significantly predicted cardiovascular reactivity in the Anger Recall condition. The practical implication for such a finding is that approaches to reduce the acute anger experience could possibly lead to lessened cardiovascular reactivity. By extension, if cardiovascular reactivity serves as a marker for development/exacerbation of cardiovascular disease, then approaches to reduce anger should also reduce the frequency of coronary events and CAD incidence. Such reasoning is not unlike that presented by previous researchers (Mittleman et al., 1995). There have been, to this point no large-scale studies focusing specifically on the use of anger-management techniques to reduce the risk for cardiovascular disease or coronary events, but one study has presented reduced anger in the context of a multifactorial risk-reduction

program in patients with cardiovascular disease (Taylor, Miller, Smith & De Busk, 1997). Their finding of overall decreases in psychological distress was not, however, related to participation in the program, nor were those findings placed in context of risk for coronary events.

From a theoretical and practical perspective, the dissertation suggests that further investigations into the psychosocial correlates of cardiovascular disease must account not only for personality and general attitudes, but must also focus on the immediate context and the individual's immediate affective state and its expression. Cardiovascular reactivity and myocardial ischemia occur most frequently in highly stressful (mentally and physically) situations, and the immediate circumstances of the environment and the individual must be considered in efforts to understand contributing factors. This supports previous epidemiological evidence regarding the importance of acute stress (Leor & Kloner, 1996; Leor, Poole, & Kloner, 1996; Meisel et al., 1991) as well as more controlled studies (see Krantz, Quigley, O'Callahan, 2001). Plainly put, acute coronary events cannot be considered without inspection of the immediate situation.

A last implication of the study findings is that defensiveness, as quantified by the Marlowe-Crowne Social Desirability Scale, does not appear to contribute to models predicting CVR and myocardial ischemia, and does not impact anger reporting. The previous studies noted earlier support this finding in part through presentation of findings related to cardiovascular reactivity and ischemia where defensiveness is presented most often in combination with some other measure such as Composite Hostility or Anger-In as related to cardiovascular endpoints. If investigators continue to show an interest in the concept of defensiveness, approaches that may possibly provide more fruitful results

could include framing defensiveness in combination with other psychosocial variables, such as the Defensive Hostility concept. In this study, Defensive Hostility, when considered with the dependent variable of stress-induced myocardial ischemia, appeared to suggest an effect not manifest by either defensiveness or hostility alone. Another option would be to develop an alternative measure of defensiveness that might be better related to expression of emotions and attitudes.

Concluding statement

The initial motivation for investigating the three hypotheses derived from the author's perception that a large proportion of those involved in the mental stress tasks were more upset and angry than they were reporting immediately after the tasks, and that evidence of this underreporting would be provided by the cardiovascular responses. Given previous research suggesting that hostility was related to both cardiovascular reactivity and myocardial ischemia, the question then arose of how trait personality measures and anger expression measures were related to each other and how they in turn were related to the cardiovascular measures of CVR and ischemia. In this study, hostility was associated with greater anger expression, but not consistently across the tasks. The findings of the study suggested that anger expression was a significant predictor of cardiovascular reactivity, but results were inconclusive regarding myocardial ischemia. Hostility and defensiveness, however, contrary to predictions, were not significantly associated in any substantial way to CVR or myocardial ischemia. It is possible that greater power would have detected a potential interactive effect, similar to previous reports of Defensive Hostility related to CVR and myocardial ischemia. It appears this study most strongly supports arguments regarding the importance of situation specificity

and acute states (such as anger and its immediate expression) over the importance of underlying personality factors (such as hostility and defensiveness) in relation to cardiovascular reactivity and myocardial ischemia. Future investigations must account for the contributions of the immediate environment and the individual's affective state just prior to and during coronary events to fully capture contributing factors to those events, just as much as they investigate acute physiological changes associated with coronary events.

TABLES

Table 1. Participant characteristics

Variable/Measure	N=59	Percentage or (Standard Deviation)
Gender		
Male (N)	52	88
Female (N)	7	12
Age (Years; mean ± SD)	61.1	(9.0)
Race		
White (N)	42	71
African-American (N)	12	20
Other (N)	4	7
Unreported (N)	1	2
Education (years)	12.5	(3.3)
Marital Status		
Married (N)	34	58
Divorced (N)	18	31
Widowed (N)	3	5
Single (N)	3	5
Unreported (N)	1	2
Living arrangement		
Alone (N)	18	31
With at least one other (N)	39	66
Unreported (N)	2	3
Work Status		
Full-time (N)	14	24
Part-time (N)	9	15
Household (N)	1	2
Unemployed (N)	3	5
Disabled (N)	5	8
Retired (N)	26	44
Unreported (N)	1	2

N= Number of participants
SD= Standard deviation

Table 2. Participant cardiovascular risk factors

Variable/Measure	N=59	Percentage or (Standard Deviation)
Body Mass Index (mean)	28.1	(5.2)
Total cholesterol (mean, mg/dl)	204.4	(39.8)
Diabetes Mellitus		
Type I (Insulin dependent; N)	11	19
Type II (Non-insulin dependent; N)	9	15
Hypertension (HTN; N)	39	66
Family History of CAD		
None (N)	24	41
First degree (N)	28	47
Second degree (N)	6	10
Unreported (N)	1	2
Family History of HTN		
None (N)	31	53
First degree (N)	23	39
Second degree (N)	4	7
Unreported (N)	1	2
Current Smoker (N)	15	25
Unreported (N)	2	3

N= number of participants
CAD= coronary artery disease
HTN= hypertension

Table 3. Cardiac history and coronary angiographic information

Variable/Measure	N=59	Percentage (or Standard Deviation)
Previously catheterized (N)	23	39
Unreported	1	2
Previous angioplasty (N)	13	22
Unreported	2	3
Previous bypass graft (N)	2	3
Previous Myocardial Infarction (N)	18	31
Unreported	1	2
Prior positive exercise stress test (N)	49	83
Unreported	10	17
NYHA Angina Class (N)		
I	11	19
II	18	31
III/IV	26	45
Unreported	4	7
Number of vessels diseased at current catheterization		
0	5	9
1	15	25
2	17	29
3	18	31
Unreported	4	7
Ejection Fraction	61.3	(12.3)

N= number of participants

Table 4. Questionnaire measures

Measure (number of items)	**N**	**Mean (SD)**
Cook Medley Total Hostility (50)	59	19.8 (9.9)
Composite Hostility (27)	59	11.9 (5.7)
Cynicism (13)	59	6.2 (3.2)
Aggressive Responding (9)	59	3.8 (2.1)
Hostile Affect (5)	58	1.9 (1.4)
Marlowe-Crowne Social Desirability (Defensiveness; 33)	56	19.5 (6.7)

N= Number of participants
SD= Standard deviation

Table 5. Anger and interest ratings

	N	**Pre-task**	**Post-task**	**Difference**	**t (sig.)**
Anger		Mean (SD)	Mean (SD)	Mean (SD)	
Math	54	1.6 (1.3)	3.8 (2.5)	2.3 (2.6)	6.51 (.001)
Anger Recall	55	1.5 (1.2)	4.0 (2.4)	2.6 (2.5)	7.52 (.001)
Stroop	45	1.5 (1.2)	2.8 (2.1)	1.2 (2.1)	3.85 (.001)
Peak	56	1.5 (1.1)	4.9 (2.1)	3.3 (2.1)	11.65 (.001)
Interest					
Math	54	4.9 (1.7)	5.4 (1.9)	0.5 (1.7)	2.12 (.039)
Anger Recall	55	5.0 (1.8)	4.9 (1.8)	-0.2 (2.0)	0.53 (.598)
Stroop	44	4.6 (2.0)	5.3 (1.9)	0.7 (2.0)	2.41 (.020)
Peak	56	4.8 (1.4)	6.1 (1.2)	1.3 (1.4)	7.20 (.001)

Ratings may range from 1 to 7, 1 representing “not at all,” 7 representing “very much.”
N = Number of participants
SD = Standard deviation

Table 6. Cardiovascular reactivity

Measure	**N***	**Pre-task**	**During Task**	**Difference ****
SBP (mmHg)		Mean (SD)	Mean (SD)	Mean (SD)
Math	57	143.4 (22.0)	167.5 (28.0)	24.1 (13.6)
Anger Recall	58	140.2 (23.8)	160.9 (27.2)	20.8 (14.0)
Stroop	45	140.6 (22.6)	162.0 (26.3)	21.4 (14.0)
DBP (mmHg)				
Math	57	77.9 (10.0)	91.0 (13.6)	13.2 (8.2)
Anger Recall	58	76.0 (10.1)	88.6 (11.7)	12.6 (7.0)
Stroop	45	78.1 (9.2)	88.8 (12.9)	10.7 (7.4)
HR (bpm)				
Math	57	64.2 (10.4)	75.9 (12.3)	11.6 (9.4)
Anger Recall	58	65.7 (11.1)	71.8 (10.6)	6.1 (4.8)
Stroop	45	64.4 (9.2)	74.9 (11.2)	10.6 (7.4)

* Numbers vary primarily due to problems with availability and operation of the computer for administering the Stroop task.

** Pre-task to task difference significant ($p < .001$) for all measures with paired samples t-test.

N= Number of participants, SD = Standard deviation, SBP= Systolic blood pressure, millimeters of mercury, DBP= Diastolic blood pressure, millimeters of mercury, HR= Heart rate, beats per minute

Table 7. Ischemia during tasks

Task	**N (RNV)**	**RNV Ischemia (%)***	**N (ECG)**	**ECG Ischemia (%)****
Math	44	9 (20.4)	56	2 (3.4)
Anger Recall	44	14 (31.8)	56	1 (1.8)
Stroop	36	9 (25)	43	1 (1.7)
Any task	47	19 (40.4)	56	3 (5.1)
Exercise	38	14 (36.8)	51	18 (35.2)

N= Number of participants

RNV = radionuclide ventriculography

ECG = Electrocardiogram

* RNV ischemia is indicated by a decrease in total wall motion score of two or more from baseline measure.

** ST segment depression of greater than 1mm during exercise test.

Table 8. Correlations between anger responses (levels and changes) by tasks, hostility measures, and subject characteristics

	Anger Aggregate Baseline	Peak Anger	Post Math Anger	Anger Change Math	Post Anger Recall Anger	Anger Change Anger Recall	Post Stroop Anger	Anger Change Stroop
Anger Aggregate Baseline	1.000**							
Peak Anger	.242	1.000**						
Post Math Anger	.342*	.711**	1.000**					
Anger Change Math	-.137	.582**	.860**	1.000**				
Post Anger Recall Anger	.203	.733**	.464**	.296*	1.000**			
Anger Change Anger Recall	-.213	.625**	.327*	.335*	.879**	1.000**		
Post Stroop Anger	.256	.414**	.358*	.213	.294	.221	1.000**	
Anger Change Stroop	-.142	.216	.102	.090	.213	.208	.848**	1.000**
Total Hostility	.283*	.111	.308*	.184	.086	.039	.228	.043
Composite Hostility	.268*	.107	.334*	.217	.056	.029	.198	-.001
Cynicism	.218	.030	.266*	.175	.011	.008	.066	-.093
Hostile Affect	.301*	.239	.404**	.225	.244	.183	.469**	.296
Aggressive Responding	.183	.062	.211	.157	-.026	-.055	.108	-.062
Age	.029	-.281*	-.053	.017	-.356**	-.411**	-.282	-.198
Education	-.229	.219	-.048	-.034	.420**	.442**	.111	.208

* = correlation is significant at < 0.05 (2-tailed), ** = correlation is significant at < 0.01 (2-tailed)
Anger Aggregate Baseline = mean of anger ratings across the three tasks; Peak Anger = highest anger rating selected from the 3 post-task anger ratings; Post Math, Post Anger Recall, and Post Stroop Anger = absolute anger reports; Anger Change Math, Anger Change Anger Recall, and Anger Change Stroop represent simple change scores, pre-post task.

Table 9a. Total Hostility to predict anger expression to Math task

Model 1. Covariates alone: $R^2 = .111$
Model 2. Covariates with Total Hostility: $R^2 = .172$ ($R^2 \Delta p = .066$)

Model	Unstandardized Coefficients		Standardized Coefficients		
	B	**Std. Error**	**Beta**	***t***	**Sig.**
Constant	.839	3.403		.247	.806
Aggregate Baseline Anger	.524	.428	.186	1.224	.227
Gender	1.189	1.242	.129	.957	.343
Age	-.020	.039	-.073	-.521	.605
Education	.050	.111	.068	.452	.653
Total Hostility	.075	.040	.298	1.879	.066

The R^2 values correspond to the percentage of variance accounted for by the model including the variables listed.
$R^2 \Delta p$ represents the p value, or significance, of the change in R^2 obtained by the model beside which it is listed compared to the previous model.
The dependent, or criterion variable, is the Likert Anger rating, post-Math task.

Table 9b. Total Hostility to predict anger expression to Anger Recall task

Model 1. Covariates alone: $R^2 = .316$
Model 2. Covariates with Total Hostility: $R^2 = .355$ ($R^2 \Delta p = .095$)

Model	Unstandardized Coefficients		Standardized Coefficients		
	B	**Std. Error**	**Beta**	***t***	**Sig.**
Constant	.772	2.954		.261	.795
Aggregate Baseline Anger	.410	.360	.152	1.140	.260
Gender	1.690	1.068	.189	1.582	.120
Age	-.069	.033	-.259	-2.071	.044
Education	.314	.099	.425	3.166	.003
Total Hostility	.058	.034	.237	1.703	.095

The R^2 values correspond to the percentage of variance accounted for by the model including the variables listed.
$R^2 \Delta p$ represents the p value, or significance, of the change in R^2 obtained by the model beside which it is listed compared to the previous model.
The dependent, or criterion variable, is the Likert Anger rating, post-Anger Recall task.

Table 9c. Total Hostility to predict anger expression to Stroop Color-Word task

Model 1. Covariates alone: $R^2 = .155$
Model 2. Covariates with Total Hostility: $R^2 = .190$ ($R^2 \Delta p = .210$)

Model	Unstandardized Coefficients		Standardized Coefficients		
	B	**Std. Error**	**Beta**	***t***	**Sig.**
Constant	1.474	3.492		.422	.675
Aggregate Baseline Anger	.604	.572	.178	1.056	.298
Gender	.905	1.088	.126	.832	.411
Age	-.046	.038	-.191	-1.213	.233
Education	.098	.105	.157	.936	.355
Total Hostility	.054	.042	.226	1.275	.210

The R^2 values correspond to the percentage of variance accounted for by the model including the variables listed.
$R^2 \Delta p$ represents the p value, or significance, of the change in R^2 obtained by the model beside which it is listed compared to the previous model.
The dependent, or criterion variable, is the Likert Anger rating, post-Stroop task.

Table 10a. Composite Hostility to predict anger expression to Math task

Model 1. Covariates alone: $R^2 = .111$
Model 2. Covariates with Composite Hostility: $R^2 = .192$ ($R^2 \Delta p = .034$)

Model	Unstandardized Coefficients		Standardized Coefficients		
	B	**Std. Error**	**Beta**	***t***	**Sig.**
Constant	.478	3.376		.142	.888
Aggregate Baseline Anger	.484	.420	.172	1.152	.255
Gender	1.080	1.231	.117	.878	.384
Age	-.021	.038	-.075	-.546	.587
Education	.071	.112	.096	.639	.526
Composite Hostility	.153	.070	.347	2.189	.034

The R^2 values correspond to the percentage of variance accounted for by the model including the variables listed.
$R^2 \Delta p$ represents the p value, or significance, of the change in R^2 obtained by the model beside which it is listed compared to the previous model.
The dependent, or criterion variable, is the Likert Anger rating, post-Math task.

Table 10b. Composite Hostility to predict anger expression to Anger Recall task

Model 1. Covariates alone: $R^2 = .316$
Model 2. Covariates with Composite Hostility: $R^2 = .350$ ($R^2 \Delta p = .118$)

Model	Unstandardized Coefficients		Standardized Coefficients		
	B	**Std. Error**	**Beta**	***t***	**Sig.**
Constant	.698	2.993		.233	.817
Aggregate Baseline Anger	.439	.358	.163	1.225	.227
Gender	1.631	1.076	.183	1.516	.136
Age	.069	.033	-.258	-2.052	.046
Education	.321	.102	.435	3.158	.003
Composite Hostility	.096	.061	.225	1.593	.118

The R^2 values correspond to the percentage of variance accounted for by the model including the variables listed.
$R^2 \Delta p$ represents the p value, or significance, of the change in R^2 obtained by the model beside which it is listed compared to the previous model.
The dependent, or criterion variable, is the Likert Anger rating, post-Anger Recall task.

Table 10c. Composite Hostility to predict anger expression to Stroop Color-Word task

Model 1. Covariates alone: $R^2 = .155$
Model 2. Covariates with Composite Hostility: $R^2 = .184$ ($R^2 \Delta p = .254$)

Model	Unstandardized Coefficients		Standardized Coefficients		
	B	**Std. Error**	**Beta**	***t***	**Sig.**
Constant	1.799	3.451		.521	.605
Aggregate Baseline Anger	.635	.572	.187	1.110	.274
Gender	.887	1.095	.123	.810	.423
Age	-.050	.038	-.206	-1.311	.198
Education	.095	.106	.152	.898	.375
Composite Hostility	.081	.070	.206	1.159	.254

The R^2 values correspond to the percentage of variance accounted for by the model including the variables listed.
$R^2 \Delta p$ represents the p value, or significance, of the change in R^2 obtained by the model beside which it is listed compared to the previous model.
The dependent, or criterion variable, is the Likert Anger rating, post-Stroop task.

Table 11a. Cynicism to predict anger expression to Math task

Model 1. Covariates alone: $R^2 = .111$
Model 2. Covariates with Cynicism: $R^2 = .155$ ($R^2 \Delta p = .124$)

Model	Unstandardized Coefficients		Standardized Coefficients		
	B	**Std. Error**	**Beta**	***t***	**Sig.**
Constant	.970	3.461		.280	.781
Aggregate Baseline Anger	.652	.414	.232	1.575	.122
Gender	1.017	1.270	.110	.800	.427
Age	-.019	.039	-.069	-.489	.627
Education	.059	.117	.080	.507	.614
Cynicism	.191	.122	.250	1.566	.124

The R^2 values correspond to the percentage of variance accounted for by the model including the variables listed.
$R^2 \Delta p$ represents the p value, or significance, of the change in R^2 obtained by the model beside which it is listed compared to the previous model.
The dependent, or criterion variable, is the Likert Anger rating, post-Math task.

Table 11b. Hostile Affect to predict anger expression to Math task

Model 1. Covariates alone: $R^2 = .104$
Model 2. Covariates with Hostile Affect: $R^2 = .230$ ($R^2 \Delta p = .008$)

Model	Unstandardized Coefficients		Standardized Coefficients		
	B	**Std. Error**	**Beta**	***t***	**Sig.**
Constant	.379	3.290		.115	.909
Aggregate Baseline Anger	.303	.423	.109	.717	.477
Gender	1.158	1.201	.127	.965	.340
Age	-.003	.038	-.011	-.079	.937
Education	.042	.107	.056	.392	.697
Hostile Affect	.746	.270	.414	2.762	.008

The R^2 values correspond to the percentage of variance accounted for by the model including the variables listed.
$R^2 \Delta p$ represents the p value, or significance, of the change in R^2 obtained by the model beside which it is listed compared to the previous model.
The dependent, or criterion variable, is the Likert Anger rating, post-Math task.

Table 11c. Hostile Affect to predict anger expression to Anger Recall task

Model 1. Covariates alone: $R^2 = .316$
Model 2. Covariates with Hostile Affect: $R^2 = .380$ ($R^2 \Delta p = .031$)

Model	Unstandardized Coefficients		Standardized Coefficients	*t*	Sig.
	B	**Std. Error**	**Beta**		
Constant	1.095	2.825		.388	.700
Aggregate Baseline Anger	.285	.363	.106	.784	.437
Gender	1.768	1.045	.198	1.693	.097
Age	-.063	.033	-.238	-1.929	.060
Education	.281	.093	.381	3.025	.004
Hostile Affect	.523	.235	.296	2.226	.031

The R^2 values correspond to the percentage of variance accounted for by the model including the variables listed.
$R^2 \Delta p$ represents the p value, or significance, of the change in R^2 obtained by the model beside which it is listed compared to the previous model.
The dependent, or criterion variable, is the Likert Anger rating, post-Anger Recall task.

Tale 11d. Hostile Affect to predict anger expression to Stroop Color-Word task

Model 1. Covariates alone: $R^2 = .149$
Model 2. Covariates with Hostile Affect: $R^2 = .311$ ($R^2 \Delta p = .006$)

Model	Unstandardized Coefficients		Standardized Coefficients	*t*	Sig.
	B	**Std. Error**	**Beta**		
Constant	1.255	3.124		.402	.690
Aggregate Baseline Anger	.160	.544	.047	.294	.770
Gender	.881	1.011	.123	.871	.389
Age	-.033	.036	-.138	-.927	.360
Education	.079	.093	.123	.849	.401
Hostile Affect	.752	.255	.465	2.947	.006

The R^2 values correspond to the percentage of variance accounted for by the model including the variables listed.
$R^2 \Delta p$ represents the p value, or significance, of the change in R^2 obtained by the model beside which it is listed compared to the previous model.
The dependent, or criterion variable, is the Likert Anger rating, post-Stroop task.

Table 12a. Correlations between cardiovascular measures (levels) and hostility measures, anger measures, and participant characteristics

	SBP Agg. Baseline	SBP Math	SBP Anger Recall	SBP Stroop	DBP Agg. Baseline	DBP Math	DBP Anger Recall	DBP Stroop	HR Agg. Baseline	HR Math	HR Anger Recall	HR Stroop
Total Hostility	-.050	-.227	-.060	-.072	-.006	-.109	.037	.028	.126	-.070	.075	-.093
Composite Hostility	-.033	-.207	-.050	-.024	-.031	-.118	.031	.020	.116	-.058	.077	-.075
Cynicism	-.122	-.264*	-.126	-.166	-.096	-.177	-.004	-.066	.064	-.066	.044	-.081
Hostile Affect	-.094	-.243	-.060	-.043	-.007	-.132	-.011	-.042	.201	.033	.132	-.120
Aggressive Responding	.160	.008	.092	.215	.058	.035	.081	.162	.081	-.070	.053	.004
Anger Agg. Baseline	.020	-.117	.059	.075	-.021	-.071	-.061	.006	-.041	-.129	-.071	-.225
Math Anger	.126	.004	.171	.180	.027	-.005	.135	.018	.379**	.154	.333*	.067
Anger Recall Anger	.123	.171	.300*	.130	.184	.250	.356**	.139	.162	.258	.301*	.032
Stroop Anger	-.079	-.091	-.004	-.048	.063	.056	-.022	-.085	.061	-.063	.013	-.159
Age	.170	.086	.058	.242	-.227	-.166	-.177	-.029	.016	-.068	-.062	.020
Education	.087	.238	.238	.179	.150	.339*	.211	.168	-.065	.275*	.005	.062

* = correlation is significant at < 0.05 (2-tailed)

** = correlation is significant at < 0.01 (2-tailed)

SBP = systolic blood pressure, DBP= diastolic blood pressure, HR = heart rate, Agg. = Aggregate

Aggregate Baseline cardiovascular measures represent the mean of the baseline measures from the 3 tasks

Anger Aggregate Baseline represents the mean of the Anger Likert pre-task ratings from the 3 tasks

Table 12b. Correlations between cardiovascular reactivity (simple change scores) and hostility measures, anger measures, and participant characteristics

	SBP Change Math	SBP Change Anger Recall	SBP Change Stroop	DBP Change Math	DBP Change Anger Recall	DBP Change Stroop	HR Change Math	HR Change Anger Recall	HR Change Stroop
Total Hostility	-.412**	-.005	-.010	-.270*	.111	.015	-.241	-.134	-.303*
Composite Hostility	-.403**	-.012	.007	-.251	.134	.029	-.209	-.106	-.257
Cynicism	-.390**	-.005	-.042	-.283*	.164	.008	-.159	-.109	-.208
Hostile Affect	-.356**	.045	-.001	-.298	.038	-.146	-.202	-.125	-.383*
Aggressive Responding	-.258	-.068	.097	-.042	.070	.150	-.185	-.021	-.116
Anger Agg. Baseline	-.214	.049	-.178	-.084	-.099	-.169	-.132	-.060	-.285
Math Anger	-.196	.114	-.088	-.008	.151	-.100	-.219	-.171	-.334*
Anger Recall Anger	.111	.363**	-.066	.151	.308*	.011	.141	.318*	.048
Stroop Anger	-.093	.160	-.061	-.039	-.054	-.246	-.143	-.109	-.262
Age	-.124	-.134	.075	.039	.044	.066	-.121	-.178	-.058
Education	.386**	.137	.041	.444**	.098	.149	.413**	.262	.364*

* = correlation is significant at < 0.05 (2-tailed)
** = correlation is significant at < 0.01 (2-tailed)
SBP = systolic blood pressure, DBP= diastolic blood pressure, HR = heart rate, Agg. = Aggregate
Aggregate Baseline cardiovascular measures represent the mean of the baseline measures from the 3 tasks
Change scores calculated as difference between cardiovascular measure during task and its preceding baseline
Anger Aggregate Baseline represents the mean of the Anger Likert pre-task ratings from the 3 tasks

Table 13a. Predictors of systolic blood pressure reactivity during Math task

Model 1. Covariates only: $R^2 = .530$
Model 2. Covariates with Composite Hostility: $R^2 = .602$ ($R^2 \Delta p = .023$)
Model 3. Covariates with Composite Hostility and Math anger rating
$R^2 = .607$ ($R^2 \Delta p = .883$)

Model	Unstandardized Coefficients		Standardized Coefficients		
	B	**Std. Error**	**Beta**	***t***	**Sig.**
Constant	8.539	21.444		.398	.693
Age	-.116	.222	-.077	-.522	.605
Gender	-5.998	6.957	-.119	-.862	.394
Education	1.136	.642	.281	1.769	.084
Diabetes	-3.562	2.822	-.198	-1.262	.214
Hypertension	-.443	4.663	-.016	-.095	.925
Number vessels diseased	.980	1.906	.072	.514	.610
Aggregate Baseline SBP	.168	.086	.281	1.953	.058
Composite Hostility	-.868	.403	-.359	-2.157	.037
Aggregate Baseline Anger	1.625	2.622	.106	.620	.539
Math Anger	-.121	.814	-.022	-.149	.883

SBP = systolic blood pressure
The R^2 values correspond to the percentage of variance accounted for by the model including the variables listed.
$R^2 \Delta p$ represents the p value, or significance, of the change in R^2 obtained by the model beside which it is listed compared to the previous model.
The dependent, or criterion variable, is the change in SBP from pre- to post-Math task.

13b. Predictors of systolic blood pressure reactivity during Anger Recall task

Model 1. Covariates only: $R^2 = .321$
Model 2. Covariates with Composite Hostility: $R^2 = .338$ ($R^2 \Delta p = .474$)
Model 3. Covariates with Composite Hostility and Anger Recall anger rating
$R^2 = .491$ ($R^2 \Delta p = .014$)

Model	Unstandardized Coefficients		Standardized Coefficients		
	B	**Std. Error**	**Beta**	***t***	**Sig.**
Constant	21.217	25.560		.830	.411
Age	.025	.279	.015	.089	.929
Gender	-12.351	8.345	-.230	-1.480	.147
Education	.387	.833	.088	.465	.645
Diabetes	-1.771	3.359	-.092	-.527	.601
Hypertension	.802	5.553	.026	.144	.886
Number vessels diseased	1.573	2.263	.109	.695	.491
Aggregate Baseline SBP	-.046	.102	-.072	-.451	.655
Composite Hostility	.036	.467	.014	.077	.939
Aggregate Baseline Anger	-.829	3.111	-.051	-.266	.791
Anger Recall Anger	2.968	1.151	.477	2.579	.014

SBP = systolic blood pressure
The R^2 values correspond to the percentage of variance accounted for by the model including the variables listed.
$R^2 \Delta p$ represents the p value, or significance, of the change in R^2 obtained by the model beside which it is listed compared to the previous model.
The dependent, or criterion variable, is the change in SBP from pre- to post-Anger Recall task.

Table 13c. Predictors of diastolic blood pressure reactivity during Anger Recall task

Model 1. Covariates only: $R^2 = .308$
Model 2. Covariates with Composite Hostility: $R^2 = .386$ ($R^2 \Delta p = .110$)
Model 3. Covariates with Composite Hostility and Anger Recall anger rating
$R^2 = .580$ ($R^2 \Delta p = .002$)

Model	Unstandardized Coefficients		Standardized Coefficients	*t*	Sig.
	B	**Std. Error**	**Beta**		
Constant	3.809	14.316		.266	.792
Age	.109	.121	.140	.905	.371
Gender	.792	4.004	.031	.198	.844
Education	.377	.382	.177	.985	.331
Diabetes	-2.755	1.490	-.298	-1.849	.072
Hypertension	3.088	2.676	.210	1.154	.255
Number vessels diseased	1.132	1.016	.164	1.114	.272
Aggregate Baseline DBP	-.180	.124	-.243	-1.457	.153
Composite Hostility	.201	.209	.163	.963	.341
Aggregate Baseline Anger	-.791	1.385	-.100	-.571	.571
Anger Recall Anger	1.747	.522	.584	3.350	.002

DBP = diastolic blood pressure
The R^2 values correspond to the percentage of variance accounted for by the model including the variables listed.
$R^2 \Delta p$ represents the p value, or significance, of the change in R^2 obtained by the model beside which it is listed compared to the previous model.
The dependent, or criterion variable, is the change in DBP from pre- to post-Anger Recall task.

Table 13d. Predictors of diastolic blood pressure reactivity during Stroop Color-Word task

Model 1. Covariates only: $R^2 = .591$
Model 2. Covariates with Composite Hostility: $R^2 = .609$ ($R^2 \Delta p = .303$)
Model 3. Covariates with Composite Hostility and Stroop anger rating
$R^2 = .692$ ($R^2 \Delta p = .044$)

Model	Unstandardized Coefficients		Standardized Coefficients		
	B	**Std. Error**	**Beta**	***t***	**Sig.**
Constant	-11.077	15.083		-.734	.468
Age	.107	.134	.127	.800	.430
Gender	-7.151	3.902	-.290	-1.833	.077
Education	.961	.399	.422	2.409	.022
Diabetes	.853	1.578	.086	.541	.593
Hypertension	2.330	2.809	.155	.830	.413
Number vessels diseased	1.678	1.076	.235	1.560	.129
Aggregate Baseline DBP	.088	.130	.118	.676	.504
Composite Hostility	.452	.239	.334	1.895	.068
Aggregate Baseline Anger	-1.940	2.243	-.160	-.865	.394
Stroop Anger	-1.077	.513	-.310	-2.099	.044

DBP = diastolic blood pressure
The R^2 values correspond to the percentage of variance accounted for by the model including the variables listed.
$R^2 \Delta p$ represents the p value, or significance, of the change in R^2 obtained by the model beside which it is listed compared to the previous model.
The dependent, or criterion variable, is the change in DBP from pre- to post-Stroop task.

Table 13e. Predictors of heart rate reactivity during Anger Recall task

Model 1. Covariates only: $R^2 = .534$
Model 2. Covariates with Composite Hostility: $R^2 = .544$ ($R^2 \Delta p = .423$)
Model 3. Covariates with Composite Hostility and Anger Recall anger rating
$R^2 = .593$ ($R^2 \Delta p = .055$)

Model	Unstandardized Coefficients		Standardized Coefficients		
	B	**Std. Error**	**Beta**	***t***	**Sig.**
Constant	10.913	8.696		1.255	.217
Age	-.089	.082	-.165	-1.081	.286
Gender	2.613	2.524	.147	1.035	.307
Education	.243	.249	.166	.977	.335
Diabetes	-.984	1.062	-.155	-.927	.359
Hypertension	.368	1.672	.036	.220	.827
Number vessels diseased	.892	.693	.188	1.288	.205
Aggregate Baseline HR	-.145	.065	-.314	-2.225	.032
Composite Hostility	.041	.143	.048	.285	.777
Aggregate Baseline Anger	-.190	.965	-.035	-.197	.845
Stroop Anger	.649	.351	.316	1.848	.072

HR = heart rate
The R^2 values correspond to the percentage of variance accounted for by the model including the variables listed.
$R^2 \Delta p$ represents the p value, or significance, of the change in R^2 obtained by the model beside which it is listed compared to the previous model.
The dependent, or criterion variable, is the change in HR from pre- to post-Anger Recall task.

14a. Ischemic status by Total Hostility, Math task

Chi-square = .005, p = .946
Odds ratio for ischemia = .950, 95% C.I. = .218 to 4.146

		Total Hostility		Total
		Low	High	
Ischemia during Math	No N	19	16	35
	%	54.3	45.7	100
	Yes N	5	4	9
	%	55.6	44.4	100
	Total N	24	20	44

14b. Ischemic status by Total Hostility, Anger Recall task

Chi-square = 2.937, p = .087
Odds ratio for ischemia = 3.109, 95% C.I. = .829 to 11.657

		Total Hostility		Total
		Low	High	
Ischemia during Anger Recall	No N	19	11	30
	%	63.3	36.7	100
	Yes N	5	9	14
	%	35.7	64.3	100
	Total N	24	20	44

14c. Ischemic status by Total Hostility, Stroop Color-Word task

Chi-square = 3.086, p = .079
Odds ratio for ischemia = 4.000, 95% C.I. = .807 to 19.818

		Total Hostility		Total
		Low	High	
Ischemia during Stroop	No N	18	9	27
	%	66.7	33.3	100
	Yes N	3	6	9
	%	33.3	66.7	100
	Total N	21	15	36

15a. Ischemic status by anger expression, Math task

Chi-square = .046, p = .830
Odds ratio for ischemia = .850, 95% C.I. = .193 to 3.739

		Anger change Math		Total
		Low	High	
Ischemia during Math	No N	17	16	34
	%	51.5	48.5	100
	Yes N	5	4	9
	%	55.6	44.4	100
Total N		22	20	42

15b. Ischemic status by anger expression, Anger Recall task

Chi-square = 1.123, p = .289
Odds ratio for ischemia = .476, 95% C.I. = .119 to 1.902

		Anger change Anger Recall		Total
		Low	High	
Ischemia during Anger Recall	No N	15	14	29
	%	51.7	48.3	100
	Yes N	9	4	13
	%	69.2	30.8	100
Total N		24	18	42

15c. Ischemic status by anger expression, Stroop Color-Word task

Chi-square = .000, p = 1.000
Odds ratio for ischemia = 1.000, 95% C.I. = .219 to 4.564

		Anger change Stroop		Total
		Low	High	
Ischemia during Stroop	No N	15	12	27
	%	55.6	44.4	100
	Yes N	5	4	9
	%	55.6	44.4	100
Total N		20	16	36

Table 16a. Correlations: Defensiveness and anger expression and interest expression (Levels)

	Anger Agg. Baseline	Math Anger	Anger Recall Anger	Stroop Anger	Interest Agg. Baseline	Math Interest	Anger Recall Interest	Stroop Interest
Defensiveness	.058	-.068	-.052	-.151	.018	.099	.131	-.135

Agg. = Aggregate.
Aggregate Anger and Interest baseline ratings were the mean of the baseline measures of the three tasks.
Ratings are the Anger and Interest Likerts obtained just following the three tasks.

Table 16b. Correlations: Defensiveness and anger expression and interest expression changes

	Anger Change Math	Anger Change Anger Recall	Anger Change Stroop	Interest Change Math	Interest Change Anger Recall	Interest Change Stroop
Defensiveness	-.071	-.159	-.077	.217	-.017	-.040

Agg. = Aggregate.
Aggregate Anger and Interest baseline ratings were the mean of the baseline measures of the three tasks.
Anger and Interest changes are the simple difference between the Likert measures taken pre- and post-task.

Table 17a. Correlations: Defensiveness and cardiovascular measures (levels)

	SBP Agg. Baseline	SBP Math	SBP Anger Recall	SBP Stroop	DBP Agg. Baseline	DBP Math	DBP Anger Recall	DBP Stroop	HR Agg. Baseline	HR Math	HR Anger Recall	HR Stroop
Defensiveness	.161	.125	.103	-.007	.020	.076	-.021	-.022	-.042	-.062	.043	.051

Agg. = Aggregate.
Aggregate baseline cardiovascular measures were the mean of the baseline measures of the three tasks.
SBP = systolic blood pressure, DBP = diastolic blood pressure, HR = heart rate
Cardiovascular measures are the mean levels of the measure during the task or baseline indicated.

Table 17b. Correlations: Defensiveness and cardiovascular reactivity

	SBP Change Math	SBP Change Anger Recall	SBP Change Stroop	DBP Change Math	DBP Change Anger Recall	DBP Change Stroop	HR Change Math	HR Change Anger Recall	HR Change Stroop
Defensiveness	.013	-.001	-.352*	.193	-.095	-.132	-.041	-.152	.154

* = Correlation is significant at < 0.05
Agg. = Aggregate.
Aggregate baseline cardiovascular measures were the mean of the baseline measures of the three tasks.
SBP = systolic blood pressure, DBP = diastolic blood pressure, HR = heart rate
Reactivity is defined as the simple pre- to post-task difference of the cardiovascular measure for the task indicated.

Table 18a. Predictors of systolic blood pressure reactivity during Anger Recall task

Model 1. Covariates only: $R^2 = .120$
Model 2. Covariates with Composite Hostility: $R^2 = .151$ ($R^2 \Delta p = .233$)
Model 3. Covariates with Composite Hostility and Defensiveness: $R^2 = .163$ ($R^2 \Delta p = .451$)
Model 4. Covariates with Composite Hostility, Defensiveness and Anger Recall anger rating: $R^2 = .302$ ($R^2 \Delta p = .010$)

Model	Unstandardized Coefficients		Standardized Coefficients		
	B	**Std. Error**	**Beta**	***t***	**Sig.**
Constant	17.749	24.788		.716	.478
Age	.148	.282	.090	.525	.602
Gender	-16.730	8.704	-.315	-1.922	.062
Education	1.461	.990	.304	1.476	.148
Hypertension	.854	5.082	.028	.168	.867
Number vessels diseased	1.197	2.355	.083	.509	.614
Aggregate Baseline SBP	-.210	.123	-.306	-1.715	.095
Composite Hostility	.461	.496	.180	.929	.359
Defensiveness	.283	.379	.121	.745	.461
Aggregate Baseline Anger	-1.444	2.854	-.089	-.506	.616
Anger Recall Anger	3.018	1.115	.474	2.708	.010

SBP = systolic blood pressure
The R^2 values correspond to the percentage of variance accounted for by the model including the variables listed.
$R^2 \Delta p$ represents the p value, or significance, of the change in R^2 obtained by the model beside which it is listed compared to the previous model.
The dependent, or criterion variable, is the change in SBP from pre- to post-Anger Recall task.

Table 18b. Predictors of systolic blood pressure reactivity during Stroop Color-Word task

Model 1. Covariates only: $R^2 = .180$
Model 2. Covariates with Composite Hostility: $R^2 = .180$ ($R^2 \Delta p = .995$)
Model 3. Covariates with Composite Hostility and Defensiveness $R^2 = .349$ ($R^2 \Delta p = .008$)
Model 4. Covariates with Composite Hostility, Defensiveness and Stroop anger rating $R^2 = .381$ ($R^2 \Delta p = .997$)

Model	Unstandardized Coefficients		Standardized Coefficients		
	B	**Std. Error**	**Beta**	***t***	**Sig.**
Constant	30.007	27.339		1.098	.281
Age	.261	.322	.157	.810	.424
Gender	-10.209	8.374	-.212	-1.219	.233
Education	-.062	.946	-.013	-.065	.949
Hypertension	-.010	5.333	.000	-.002	.998
Number vessels diseased	3.449	2.502	.246	1.379	.179
Aggregate Baseline SBP	.109	.124	.162	.877	.388
Composite Hostility	-.304	.537	-.115	-.565	.576
Defensiveness	-1.150	.405	-.507	-2.841	.008
Aggregate Baseline Anger	-5.782	4.842	-.244	-1.194	.242
Stroop Anger	-.004	1.107	-.001	-.004	.997

SBP = systolic blood pressure
The R^2 values correspond to the percentage of variance accounted for by the model including the variables listed.
$R^2 \Delta p$ represents the p value, or significance, of the change in R^2 obtained by the model beside which it is listed compared to the previous model.
The dependent, or criterion variable, is the change in SBP from pre- to post-Stroop task.

Table 18c. Predictors of diastolic blood pressure reactivity during Anger Recall task

Model 1. Covariates only: $R^2 = .083$
Model 2. Covariates with Composite Hostility: $R^2 = .156$ ($R^2 \Delta p = .070$)
Model 3. Covariates with Composite Hostility and Defensiveness $R^2 = .162$ ($R^2 \Delta p = .587$)
Model 4. Covariates with Composite Hostility, Defensiveness and Anger Recall anger rating $R^2 = .324$ ($R^2 \Delta p = .006$)

Model	Unstandardized Coefficients		Standardized Coefficients		
	B	**Std. Error**	**Beta**	***t***	**Sig.**
Constant	2.037	15.025		.136	.893
Age	.114	.125	.145	.912	.368
Gender	1.861	4.397	.073	.423	.675
Education	.387	.429	.168	.901	.373
Hypertension	.413	2.419	.028	.171	.865
Number vessels diseased	1.294	1.109	.188	1.167	.251
Aggregate Baseline DBP	-.137	.131	-.187	-1.040	.305
Composite Hostility	.284	.229	.232	1.240	.223
Defensiveness	-.117	.176	-.105	-.664	.511
Aggregate Baseline Anger	-1.560	1.341	-.200	-1.163	.252
Anger Recall Anger	1.509	.519	.496	2.906	.006

DBP = diastolic blood pressure
The R^2 values correspond to the percentage of variance accounted for by the model including the variables listed.
$R^2 \Delta p$ represents the p value, or significance, of the change in R^2 obtained by the model beside which it is listed compared to the previous model.
The dependent, or criterion variable, is the change in DBP from pre- to post-Anger Recall task.

Table 18d. Predictors of diastolic blood pressure reactivity during Stroop Color-Word task

Model 1. Covariates only: $R^2 = .347$
Model 2. Covariates with Composite Hostility: $R^2 = .369$ ($R^2 \Delta p = .302$)
Model 3. Covariates with Composite Hostility, and Defensiveness $R^2 = .410$ ($R^2 \Delta p = .152$)
Model 4. Covariates with Composite Hostility, Defensiveness and Stroop anger rating $R^2 = .528$ ($R^2 \Delta p = .030$)

Model	Unstandardized Coefficients		Standardized Coefficients		
	B	**Std. Error**	**Beta**	***t***	**Sig.**
Constant	-10.316	14.634		-.705	.486
Age	.149	.132	.174	1.126	.270
Gender	-4.572	4.070	-.186	-1.123	.270
Education	.882	.390	.372	2.261	.031
Hypertension	3.215	2.476	.213	1.298	.204
Number vessels diseased	2.280	1.138	.318	2.004	.055
Aggregate Baseline DBP	.117	.127	.156	.918	.366
Composite Hostility	.273	.240	.202	1.138	.264
Defensiveness	-.334	.183	-.287	-1.827	.078
Aggregate Baseline Anger	-1.788	2.139	-.148	-.836	.410
Stroop Anger	-1.126	.493	-.322	-2.285	.030

DBP = diastolic blood pressure
The R^2 values correspond to the percentage of variance accounted for by the model including the variables listed.
$R^2 \Delta p$ represents the p value, or significance, of the change in R^2 obtained by the model beside which it is listed compared to the previous model.
The dependent, or criterion variable, is the change in DBP from pre- to post-Stroop task.

Table 19a. Ischemic status by Defensiveness, Math task

Chi-square = .655, p = .418
Odds ratio for ischemia = 1.882, 95% C.I. = .402 to 8.824

		Defensiveness		Total
		Low	High	
Ischemia during Math	No N	16	17	33
	%	48.5	51.5	100
	Yes N	3	6	9
	%	33.3	66.7	100
Total N		19	23	42

Table 19b. Ischemic status by Defensiveness, Anger Recall task

Chi-square = .006, p = .936
Odds ratio for ischemia = .948, 95% C.I. = .255 to 3.525

		Defensiveness		Total
		Low	High	
Ischemia during Math	No N	13	16	29
	%	44.8	55.2	100
	Yes N	6	7	13
	%	46.2	53.8	100
Total N		19	23	42

Table 19c. Ischemic status by Defensiveness, Stroop Color-Word task

Chi-square = .001, p = .982
Odds ratio for ischemia = .982, 95% C.I. = .212 to 4.553

		Defensiveness		Total
		Low	High	
Ischemia during Math	No N	11	14	25
	%	44.0	56.0	100
	Yes N	4	5	9
	%	44.4	55.6	100
Total N		15	19	34

Table 20. Correlations: Composite Hostility, Defensiveness, Composite Hostility X Defensiveness Interaction

	Composite Hostility	Defensiveness	Composite Hostility X Defensiveness
Composite Hostility	1.000	-.359**	.712**
Defensiveness	-.359**	1.000	.305*
Composite Hostility X Defensiveness	.712**	.305*	1.000

* = Correlation is significant at $p < .05$ level.
**= Correlation is significant at $p < .01$ level.

Table 21. Participants' ischemic status for any task based on Defensive Hostility classification
($\chi^2 = 3.93$, $p = .269$)

	Ischemia (%)	No ischemia (%)	Total N
Low Hostile	4 (44)	5 (56)	9
High Hostile	4 (31)	9 (69)	13
Defensive	5 (31)	11 (69)	16
Defensive Hostile	5 (71)	2 (29)	7

Ischemic status for any task indicates the participant was ischemic during at least one of the three mental stress tasks.

Table 22a. Ischemia status and cardiovascular reactivity, Math task

	Ischemia Negative N= 33	Ischemia Positive N=9	Overall Mean Change
SBP Mean Change (S.D.)	25 (13)	23 (13)	24 (13)
DBP Mean Change (S.D.)	15 (8)	13 (8)	14 (8)
HR Mean Change (S.D.)	12 (10)	10 (6)	12 (9)

SBP= systolic blood pressure, DBP= diastolic blood pressure, HR = heart rate, S.D. = standard deviation

Changes are calculated as the simple difference of the cardiovascular measures baseline and during task.

Table 22b. Ischemia status and cardiovascular reactivity, Anger Recall task

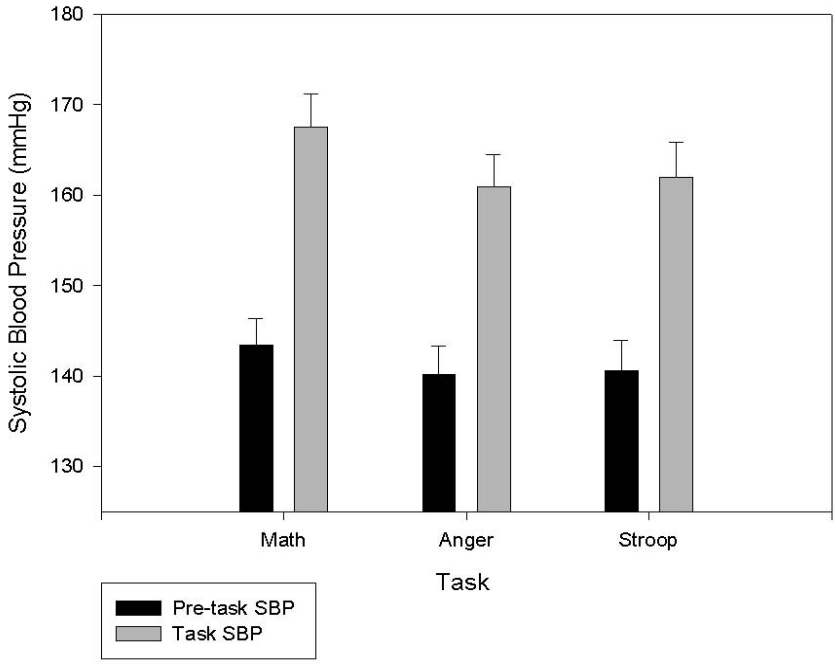
	Ischemia Negative N= 30	Ischemia Positive N=14	Overall Mean Change
SBP Mean Change (S.D.)	22 (14)	19 (13)	21 (14)
DBP Mean Change (S.D.)	12 (8)	12 (5)	12 (7)
HR Mean Change (S.D.)	6 (5)	7 (6)	6 (5)

Table 22c. Ischemia status and cardiovascular reactivity, Stroop task

	Ischemia Negative N= 25	Ischemia Positive N=9	Overall Mean Change
SBP Mean Change (S.D.)	23 (14)	26 (15)	24 (14)
DBP Mean Change (S.D.)	11 (9)	9 (8)	11 (8)
HR Mean Change (S.D.)	11 (8)	8 (6)	11 (7)

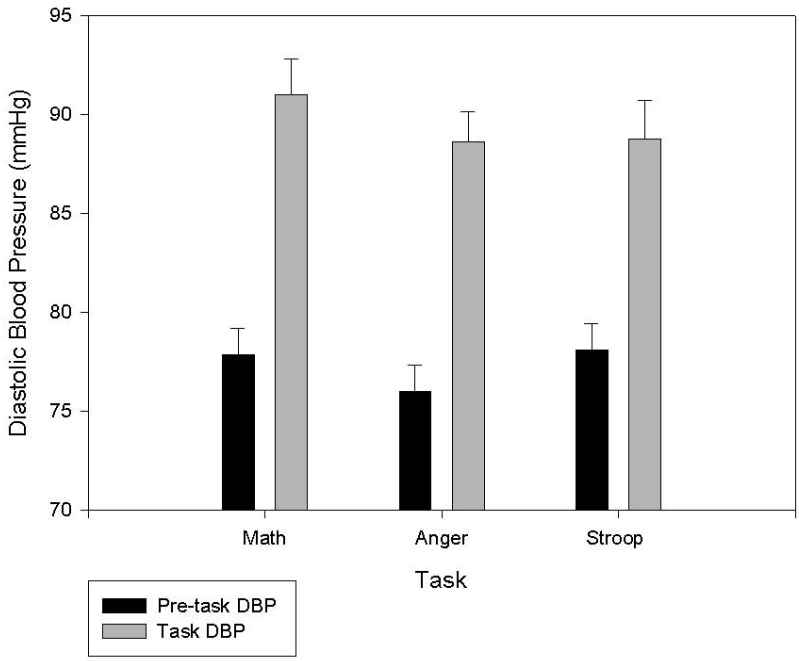
FIGURES

Figure 1. Systolic Blood Pressure Levels Across Tasks



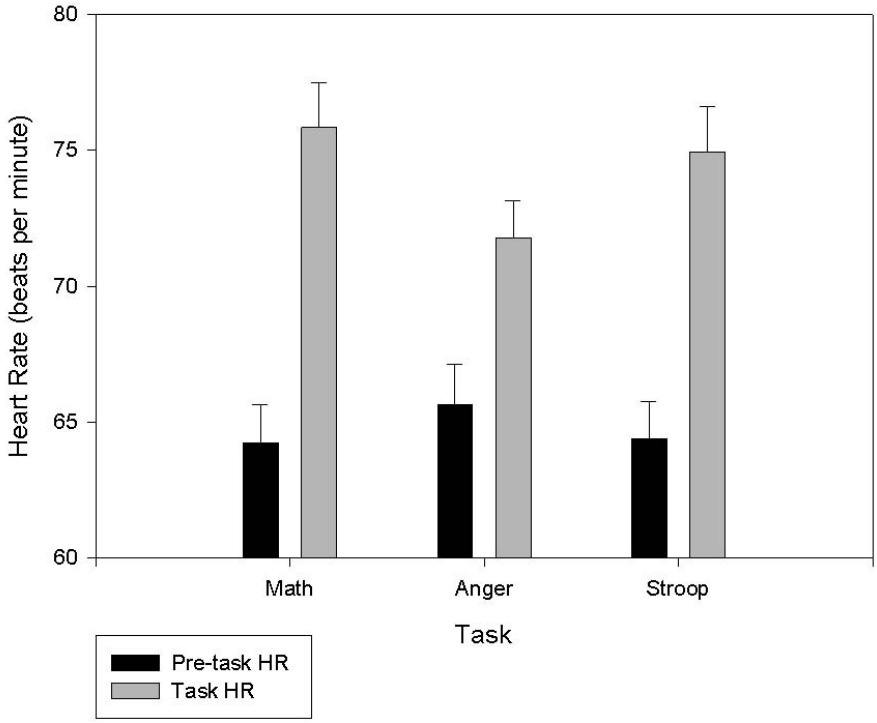
Error bars represent standard error

Figure 2. Diastolic Blood Pressure Levels by Task



Error bars represent standard error

Figure 3. Heart Rate Levels by task



Error bars represent standard error

Figure 4. Presence of ischemia by Total Hostility, Anger Recall task

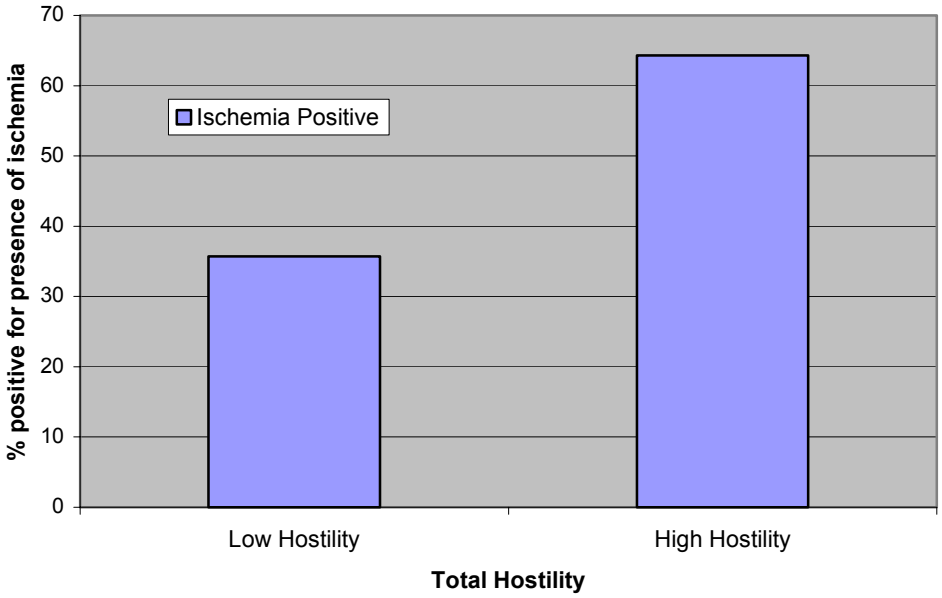


Figure 5. Presence of myocardial ischemia by Total Hostility, Stroop Color-Word task

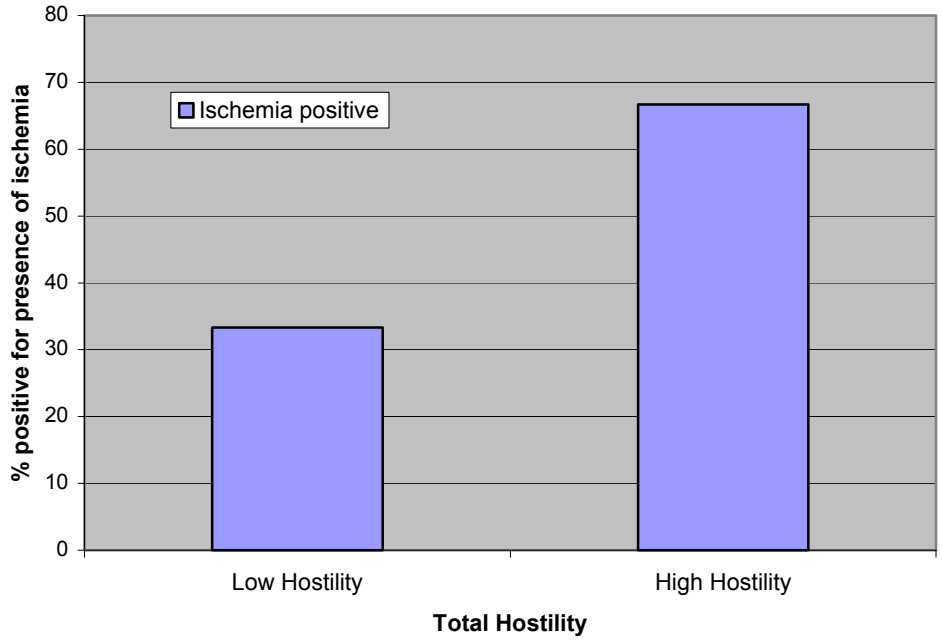
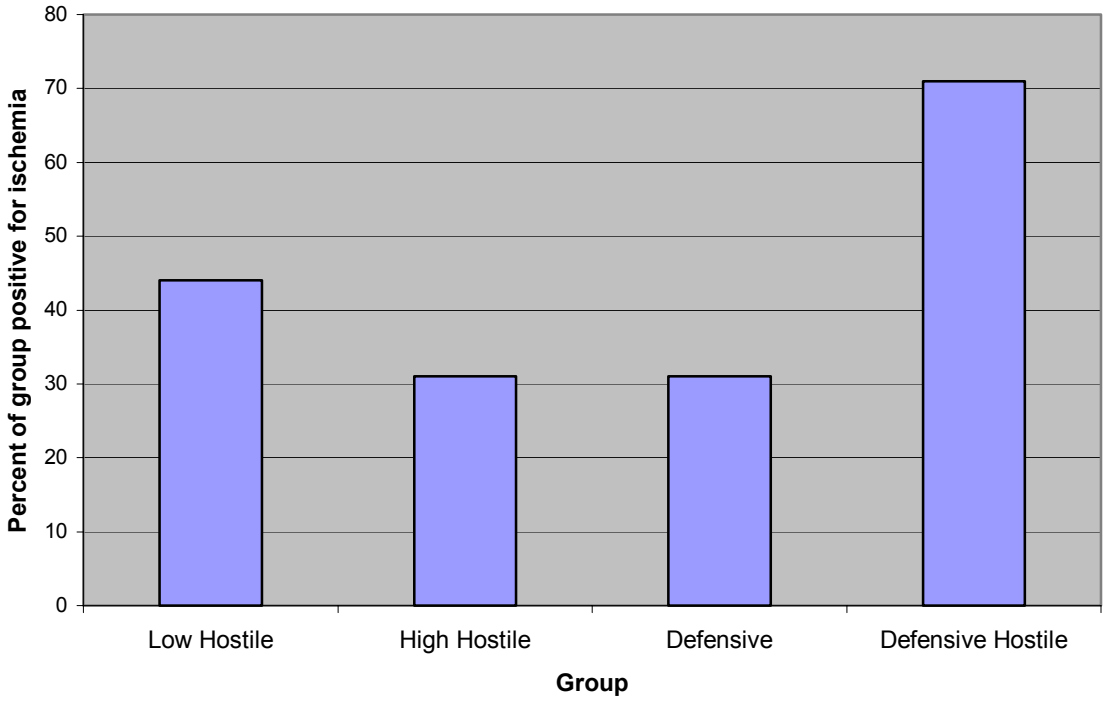


Figure 6. Presence of myocardial ischemia by Defensive Hostility Group, All tasks



Low Hostile: Low Composite Hostility, Low Defensiveness
High Hostile: High Composite Hostility, Low Defensiveness
Defensive: Low Composite Hostility, High Defensiveness
Defensive Hostile: High Composite Hostility, High Defensiveness

APPENDIX

time: _____:_____

rest # 1 (after rest 1)

Instruction: Below are words which describe the feelings people have. Please read each one carefully and rate how much you have had that feeling during the past 5 minutes.

	not at all						very much
anxious	(1)	(2)	(3)	(4)	(5)	(6)	(7)
frustrated	(1)	(2)	(3)	(4)	(5)	(6)	(7)
irritated	(1)	(2)	(3)	(4)	(5)	(6)	(7)
tired	(1)	(2)	(3)	(4)	(5)	(6)	(7)
challenged	(1)	(2)	(3)	(4)	(5)	(6)	(7)
depressed	(1)	(2)	(3)	(4)	(5)	(6)	(7)
interested	(1)	(2)	(3)	(4)	(5)	(6)	(7)
angry	(1)	(2)	(3)	(4)	(5)	(6)	(7)
chest pain	(1)	(2)	(3)	(4)	(5)	(6)	(7)

time: _____:_____

mental stress # 1 _____

Instruction: Below are words which describe the feelings people have. Please read each one carefully and rate how much you have had that feeling during the past 5 minutes.

	not at all						very much
anxious	(1)	(2)	(3)	(4)	(5)	(6)	(7)
frustrated	(1)	(2)	(3)	(4)	(5)	(6)	(7)
irritated	(1)	(2)	(3)	(4)	(5)	(6)	(7)
tired	(1)	(2)	(3)	(4)	(5)	(6)	(7)
challenged	(1)	(2)	(3)	(4)	(5)	(6)	(7)
depressed	(1)	(2)	(3)	(4)	(5)	(6)	(7)
interested	(1)	(2)	(3)	(4)	(5)	(6)	(7)
angry	(1)	(2)	(3)	(4)	(5)	(6)	(7)
chest pain	(1)	(2)	(3)	(4)	(5)	(6)	(7)

Cook-Medley Scale

Read each statement and decide whether each is true as applied to you or false as applied to you. If a statement is true or mostly true, as applied to you, circle the **T** following the statement. If a statement is false or not usually true, as applied to you, circle the **F** following the statement. If a statement does not apply to you, or if it is something you do not know about, make no mark. Remember to give your own opinion of yourself. Do not leave any spaces blank if you can avoid it.

1.	When someone does me wrong I feel I should pay him back if I can, just for the principle of the thing.	T	F
2.	I prefer to pass by school friends, or people I know but have not seen for a long time, unless they speak to me first.	T	F
3.	I have often had to take orders from someone who did not know as much as I did.	T	F
4.	I think a great many people exaggerate their misfortune in order to gain the sympathy and help of others.	T	F
5.	It takes a lot of argument to convince most people of the truth.	T	F
6.	I think most people would lie to get ahead	T	F
7.	Someone has it in for me.	T	F
8.	Most people are honest chiefly through fear of being caught.	T	F
9.	Most people will use somewhat unfair means to gain profit or an advantage rather than to lose it.	T	F
10.	I commonly wonder what hidden reason another person may have for doing something nice for me.	T	F
11.	It makes me impatient to have people ask for my advice or otherwise interrupt me when I am working on something important.	T	F
12.	I feel that I have often been punished without cause.	T	F
13.	I am against giving money to beggars.	T	F

14.	Some of my family have habits that bother and annoy me very much.	T	F
15.	No one cares much what happens to you.	T	F
16.	My relatives are nearly all in sympathy with me.	T	F
17.	My way of doing things is apt to be misunderstood by others.	T	F
18.	I don't blame anyone for trying to grab everything he can get in this world.	T	F
19.	Most people make friends because friends are likely to be useful to them.	T	F
20.	I am sure I am being talked about.	T	F
21.	I am likely not to speak to people until they speak to me.	T	F
22.	Most people inwardly dislike putting themselves out to help other people.	T	F
23.	I tend to be on my guard with people who are somewhat more friendly than I had expected.	T	F
24.	I have sometimes stayed away from another person because I feared doing or saying something that I might regret afterwards.	T	F
25.	People often disappoint me.	T	F
26.	I like to keep people guessing what I'm going to do next.	T	F
27.	I frequently ask people for advice.	T	F
28.	I am not easily angered.	T	F
29.	I have often met people who were supposed to be experts who were no better than I.	T	F
30.	I would certainly enjoy beating a crook at his own game.	T	F

31.	It makes me feel like a failure when I hear of the success of someone I know well.	T	F
32.	I have at times had to be rough with people who were rude or annoying.	T	F
33.	People generally demand more respect for their own rights than they are willing to allow for others.	T	F
34.	There are certain people whom I dislike so much that I am inwardly pleased when they are catching it for something they have done.	T	F
35.	I am often inclined to go out of my way to win a point with someone who has opposed me.	T	F
36.	I am quite often not in on the gossip and talk of the group I belong to.	T	F
37.	The man who had the most to do with me when I was a child (such as my father, stepfather, etc.) was very strict with me.	T	F
38.	I have often found people jealous of my good ideas, just because they had not thought of them first.	T	F
39.	When a man is with a woman he is usually thinking about things related to her sex.	T	F
40.	I do not try to cover up my poor opinion or pity of a person so that he won't know how I feel.	T	F
41.	I have frequently worked under people who seem to have things arranged so that they get credit for good work but are able to pass off mistakes onto those under them.	T	F
42.	I strongly defend my own opinions as a rule.	T	F
43.	People can pretty easily change me even though I thought that my mind was already made up on a subject.	T	F
44.	Sometimes I am sure that other people can tell me what I am thinking.	T	F
45.	A large number of people are guilty of bad sexual conduct.	T	F

46.	When I take a new job, I like to be tipped off on who should be gotten next to.	T	F
47.	I have often felt that strangers were looking critically at me	T	F
48.	I can be friendly with people who do things which I consider wrong.	T	F
49.	It is safer to trust nobody.	T	F
50.	I do not blame a person for taking advantage of someone who lays himself open to it.	T	F

Taylor/Crowne-Marlowe Inventory

Please read each statement and decided whether it is mostly true as applied to you or mostly false. Please put an "X" in the appropriate column to the left of each item. Answer "TRUE" to positively stated questions if they are true as often or more often than stated. For example, answer "TRUE" to "Occasionally I play poker" if you play poker occasionally or more often.

	TRUE	FALSE
1. I find it hard to keep my mind on a task or job.	_____	_____
2. I am sometimes irritated by people who ask favors of me.	_____	_____
3. I am happy most of the time.	_____	_____
4. Before voting I thoroughly investigate the qualifications of all the candidates.	_____	_____
5. I believe I am no more nervous than most others.	_____	_____
6. I sometimes think when people have a misfortune they only got what they deserved.	_____	_____
7. I am more sensitive than most other people.	_____	_____
8. I like gossip at times.	_____	_____
9. On occasion I have had doubts about my ability to succeed in life.	_____	_____
10. There have been occasions when I took advantage of someone.	_____	_____
11. I am a high-strung person.	_____	_____
12. I have never intensely disliked someone.	_____	_____
13. I cannot keep my mind on one thing.	_____	_____
14. I never make a long trip without checking the safety of my car.	_____	_____
15. I have periods of such great restlessness that I cannot sit long in a chair.	_____	_____
16. I am always courteous, even to people who are disagreeable.	_____	_____
17. On a few occasions, I have given up doing something because I thought too little of my ability.	_____	_____
18. I am always careful about my manner of dress.	_____	_____

	TRUE	FALSE
19. At times I think I am no good at all.	_____	_____
20. I have never felt that I was punished without cause.	_____	_____
21. When I don't know something I don't at all mind admitting it.	_____	_____
22. I am usually calm and not easily upset.	_____	_____
23. I never resent being asked to return a favor.	_____	_____
24. I am not unusually self-conscious.	_____	_____
25. I sometimes try to get even, rather than forgive and forget.	_____	_____
26. If I could get into a movie without paying and be sure I was not seen, I would probably do it.	_____	_____
27. I work under a great deal of tension.	_____	_____
28. I have never deliberately said something that hurt someone's feelings.	_____	_____
29. I can remember "playing sick" to get out of something.	_____	_____
30. I am inclined to take things hard.	_____	_____
31. I sometimes feel resentful when I don't get my way.	_____	_____
32. Life is a strain for me much of the time.	_____	_____
33. No matter who I'm talking to, I'm always a good listener.	_____	_____
34. I certainly feel useless at times.	_____	_____
35. I always try to practice what I preach.	_____	_____
36.There have been times when I was quite jealous of the good fortune of others.	_____	_____
37. I sometimes feel that I am about to go to pieces.	_____	_____
38. I have never been irked when people expressed ideas very different from my own.	_____	_____
39. My table manners at home are as good as when I eat out in the restaurant.	_____	_____
40. There have been occasions when I felt like smashing things.	_____	_____
41. I have sometimes felt that difficulties were piling up so high that I could not overcome them.	_____	_____

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	TRUE	FALSE
42. I never hesitate to go out of my way to help someone in trouble.	_____	_____
43. It is sometimes hard for me to go on with my work if I am not encouraged	_____	_____
44. At times I have really insisted on having things my own way.	_____	_____
45. I feel anxiety about something or someone almost all the time.	_____	_____
46. I am always willing to admit it when I make a mistake.	_____	_____
47. There have been times when I felt like rebelling against people in authority even though I knew they were right.	_____	_____
48. I frequently find myself worrying about something.	_____	_____
49. I have almost never felt the urge to tell someone off.	_____	_____
50. I shrink from facing a crisis or difficulty.	_____	_____
51. I don't find it particularly difficult to get along with loud-mouthed, obnoxious people.	_____	_____
52. I am certainly lacking in self-confidence.	_____	_____
53. I would never think of letting someone else be punished for my wrong-doing.	_____	_____

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